The electromyographic threshold in children: Differences in neuromuscular activation during progressive exercise between boys and men

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Abstract

The electromyographic threshold (EMG_{Th}), defined as an upward inflexion in the rising EMG signal during progressive exercise, is thought to reflect the onset of increased type-II MU recruitment. The study's objective was to compare the relative exercise intensity at which the EMG_{Th} occurs in boys *vs.* men.

Participants included 21 men (23.4±4.1 yrs) and 23 boys (11.1±1.1 yrs). Ramped cycle-ergometry was conducted to volitional exhaustion with surface EMG recorded from the vastus lateralis muscles. The EMG_{Th} was mathematically determined using a composite of both legs.

EMG_{Th} was detected in 95.2% of the men and in 78.3% of the boys ($\chi^2_{(1,\ n=44)} = 2.69$, $p = .10$). The boys' EMG_{Th} was significantly higher than the men's (86.4±9.6 vs. 79.7±10.0% of peak power-output at exhaustion; $p < .05$).

These findings suggest that boys activate their type-II MUs to a lesser extent than men during progressive exercise and support the hypothesis of differential child–adult MU activation.

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Abbreviations / Key Terms

- EMG_{Th} = electromyographic (EMG) threshold
- ICC = Intraclass Correlation Coefficient
- MFCV = muscle fibre conduction velocity
- MU = motor unit
- MVC = maximal voluntary contraction
- VO_2pk = peak oxygen consumption (VO_2)
- PO_2pk = peak aerobic power *i.e.*, the power output (Watt) corresponding to VO_2pk
- PHV = peak height velocity
- RMS = root mean square
- $VO_2pk/kgBW$ = specific VO_2pk; VO_2pk divided by body weight in kg
- P_{max} = peak power output (Watt) achieved in the EMG_{Th} test
- $P_{max}/kgBW$ = specific P_{max}; P_{max} divided by body weight in kg

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Background

Children are physiologically and functionally different from adults. In particular, children show a number of characteristic differences from adults in terms of muscular performance during exercise, metabolic responses to exercise, and neuromuscular function (Appendix A, Table 4, Dotan et al. 2012).

Performance-related child–adult differences

Even after accounting for differences in body dimensions, children demonstrate lower maximal isometric strength (Falk et al. 2009b), lower short-term power (Van Praagh & Dore 2002; Beneke et al. 2007; Bedu et al. 1991; Dotan et al. 2003), and slower force kinetics relative to adults (Going et al. 1987; Asai & Aoki 1996; Cohen et al.2010; Falk et al. 2009a, b). For example, after controlling for upper arm cross-sectional area and peak electromyographic activity, boys generated significantly lower peak torque than men during maximal isometric elbow flexion (Falk et al. 2009b). Maximal short-term power output, as assessed, for example, using the Wingate-Anaerobic-Test, has also been shown to be significantly lower in children than in both adults and adolescents (Van Praagh & Dore 2002; Beneke et al. 2007; Bedu et al. 1991; Dotan et al. 2003).

Children have also been shown to have lower peak rates of force development during both maximal isometric contractions (Going et al. 1987; Asai & Aoki 1996; Cohen et al.2010; Falk et al. 2009a, b) and twitch contractions (Grosset et al. 2005). For example, it took boys nearly 50% longer than men to develop 80% of their maximal torque during maximal isometric knee extensions (Dotan et al. 2013). Children further show different force-velocity relationships from adults (Asai & Aoki 1996; Barrett &

Harrison 2002). For example, at any given force level, contraction velocity during elbow flexion was found to be ~35% lower in boys than in men (Asai & Aoki 1996).

The increase in isokinetic torque production, measured during concentric and eccentric quadriceps contractions, was also shown to be lower in boys compared with adolescents (Seger & Thorstensson 2000). This age-related difference in torque production was greater at higher isokinetic contraction velocities, indicating both higher contractile velocity and higher torque production in the adolescents compared with the boys (Seger & Thorstensson 2000).

Children also show enhanced muscular endurance compared with adults, as evidenced by their lower muscle fatigability during both dynamic (Armatas et al. 2010; Kotzamanidou et al. 2005; Paraschos et al. 2007; Zafeiridis et al. 2005) and isometric (Halin et al. 2003) contractions. For example, during repeated maximal, dynamic knee extensions, boys completed ~70% of their total number of repetitions before torque declined to below 50% of its initial value, whereas men exhibited a comparable decline following only ~20% of total repetitions (Armatas et al. 2010).

Metabolic-related child–adult differences

Additional child–adult differences in metabolic responses to exercise include a lower peak blood-lactate concentration ([La]pk) (Dotan et al. 2003), higher lactate (Simon et al. 1981; Tanaka & Shindo 1985) and ventilatory thresholds (Klentoru et al. 2006), a higher intracellular inorganic phosphate/phosphocreatine threshold (Willcocks et al. 2010), enhanced intracellular phosphocreatine recovery (Taylor et al. 1997; Willcocks et al. 2010), greater reliance on fat metabolism (Riddell et al. 2008), and faster VO_2 kinetics in children relative to adults (Fawkner & Armstrong 2004; Springer et al. 1991;

Williams et al. 2001)). Following the 30-second Wingate-Anaerobic-Test, [La]pk was shown to be 52% lower in prepubertal boys compared with men (Dotan et al. 2003), a finding consistent with several other studies reporting a lesser lactate response to exercise in children relative to adults (Beneke et al. 2005; Ratel et al. 2002a,b; Hebestreit et al. 1996; Zafeiridis et al. 2005). The intracellular threshold of the inorganic phosphate/phosphocreatine ratio has been shown to occur at ~20% higher relative power output in children relative to adults (Willcocks et al. 2010), while phosphocreatine resynthesis following graded calf-muscle exercise was also found to occur faster in children than in adults (Taylor et al. 1997). It has further been demonstrated that children reach a given percentage of their VO_2 response to a given exercise much faster than both adolescents and adults (Fawkner & Armstrong 2004; Springer et al. 1991; Williams et al. 2001), likely reflecting faster phase-II pulmonary VO_2 kinetics (*i.e.*, the slow component of the VO_2 response) in children compared with adults (Fawkner & Armstrong 2004; Williams et al. 2001; Fawkner et al. 2002).

Fat oxidation rates during graded exercise testing were found to peak at 55% of VO_2pk in prepubertal boys and at ~30% VO_2pk in men, indicating a higher reliance on fat metabolism towards higher exercise intensities in the children. Furthermore, as the boys matured over the three-year longitudinal study, peak fat oxidation rates occurred at progressively lower percentages of VO_2pk (Riddell et al. 2008).

Children's recovery from high-intensity, short-term exercise in has also been found to be faster than in adults, likely stemming from metabolic differences such as faster energy substrate replenishment and acid-base rebalance in children relative to adults (Falk & Dotan 2006). Higher plasma and intramuscular pH, smaller changes in

plasma strong ion concentrations, and smaller increases in plasma pCO_2 during repeated high-intensity cycling sprints have all been shown in children, relative to adults (Zanconato et al. 1993; Hebestreit et al. 1996; Ratel et al. 2002a; Dotan et al. 2003). When monitored post exercise, these metabolic changes appeared earlier and diminished more rapidly in children compared with adults (Zanconato et al. 1993; Hebestreit et al. 1996; Ratel et al. 2002a).

Threshold-related child–adult differences

The blood lactate threshold, which reflects the onset of accelerated blood-lactate accumulation during progressively-intensifying exercise, has been found to consistently occur at a higher percentage of VO_2pk in children than in both adolescents and adults (Simon et al. 1981; Tanaka & Shindo 1985; Pfitzinger & Freedson 1997). Ventilatory threshold is the exercise intensity at which ventilation (VE) increases disproportionately relative to VO_2, reflecting increased blood pCO_2 and acidity. It too has been shown to occur at higher relative exercise intensities in children: ~65% VO_2pk in boys versus ~58% VO_2pk in men (Klentrou et al. 2006).

Neuromuscular-related child–adult differences

The differences in neuromuscular activation between children and adults are perhaps the most intriguing given that differential neuromuscular activation patterns could potentially underlie all of the above child–adult differences in muscular performance and metabolic responses to exercise. Lower muscle activation in children relative to adults has been repeatedly demonstrated using the interpolated-twitch technique, whereby an electrical or magnetic stimulus is applied to a muscle or motor neuron during maximal voluntary contraction to induce a twitch that presumably recruits

all motor units, and then measure the additional force beyond MVC (Belanger & McComas 1989; Blimkie 1989; Grosset et al. 2008; Paasuke et al. 2000). Significantly lower motor-unit activation of the plantar flexor (Belanger & McComas 1989, Grosset et al. 2008), knee extensor (Blimkie 1989; O'Brien et al. 2009, 2010), and adductor pollicis (Martin et al. 2013) muscles has been found in children compared with adults.

Children are also thought to have a lower rate of muscle activation as evidenced by their lower Q_{30} index values relative to adults (Cohen et al. 2010; Falk et al. 2009a). The Q_{30} represents the rate of rise in the integral of the rectified surface EMG signal during the first 30ms of EMG activity and has been suggested to reflect the rate of motor unit activation (Gottlieb et al. 1989). Compared with adults, children further show a longer electromechanical delay (Asai & Aoki 1996; Cohen et al. 2010; Falk et al. 2009a, b). The electromechanical delay represents the short period of latency between neuromuscular activation (EMG onset) and force production by the muscle (force onset) (Asai & Aoki 1996; Cohen et al. 2010; Falk et al. 2009a, b). However, the difference in electromechanical delay between children and adults is likely mainly due to lower musculo-tendinous stiffness in children, rather than differential motor-unit activation (Dotan et al. 2012). In addition, children have been found to display a lower mean power frequency (MPF) during sustained maximal contraction (Halin et al. 2003), and more importantly a smaller decrease in MPF (*i.e.* leftward shift towards lower frequencies in the MPF distribution curve) than adults during isometric fatigue tests (Halin et al. 2003; Armatas et al. 2010). The MPF is thought to reflect the size and frequency of motor-unit action potentials and to vary with changing motor neuron firing rates (Kamen & Gabriel 2010).

Possible explanations

Several factors may contribute to the explanation of the above observed child–adult differences: anatomic, metabolic, and functional (Dotan et al. 2012). Briefly, these possible explanations include children's lower relative muscle size, lower anaerobic and higher oxidative muscle metabolic profile, higher agonist-antagonist co-activation during muscle contractions, lower intra-muscular synchronization, lower overall volitional muscle activation, and lower type-II muscle-fibre composition (Dotan et al. 2012). While each of these factors can partially account for many or fully account for a number of the observed differences, only two factors can potentially provide an explanation, either full or partial, for each of the observed child–adult differences in muscular performance and metabolic response to exercise outlined above.

The first of these is the possibility that children have a lower type-II muscle-fibre composition compared with adults. Muscle-fibre type composition data for children is limited due to the ethical constraints of taking muscle biopsies from children. Nevertheless, three studies have suggested age-related increases in the percentage of type-II fibre types (Jansson 1996; Lexell et al. 1992; Glenmark et al. 1992), from childhood through adolescence, and into adulthood. In conflict with this view, various other studies, including some examining contractile characteristics as an indirect measure of muscle composition, have suggested comparable fibre-type composition in children and adults (Bell et al. 1980; Brooke & Engel 1969; Vogler & Bove 1985; Belanger & McComas 1989; Davies 1983, 1985; McComas et al. 1973; Paasuke et al. 2000).

The second explanation, termed the differential motor-unit activation hypothesis, suggests that children activate their fast-twitch, glycolytic, higher threshold type-II

motor-units to a lesser extent than adults, activating their higher endurance, slow-twitch, oxidative, lower threshold type-I motor-units proportionally more during muscular contraction and exercise (Asmussen & Heeboll-Nielsen 1955; Asai & Aoki 1996; Belanger & McComas 1989; Blimkie 1989; Falk et al. 2009a, b; Dotan et al. 2012). Such differential motor-unit activation between children and adults could be caused by some combination of varying patterns of motor-unit recruitment or rate-coding. Motor-unit recruitment describes whether individual motor-units are activated during contraction, while rate-coding refers to the firing rate of recruited motor-units. Whatever the case, lower type-II motor-unit use may underlie all of the child–adult differences discussed above.

In humans, three distinct skeletal-muscle fibre types have traditionally been described, based on histochemical staining for myosin ATPase (Staron 1997; Bottinelli & Reggiani 2000), myosin heavy chain (MHC) isoform identification, and biochemical identification of metabolic enzymes (Scott et al. 2001). Myosin ATPase staining is used to separate fibre types into fast and slow twitch. Fibres are further divided into oxidative or glycolytic based on the activity and biochemical identification of metabolic enzymes. Type-I fibres are characterized as slow-twitch muscle fibres (Bottinelli & Reggiani 2000), showing lower rates of myosin ATPase hydrolysis (Taylor et al. 1974) and thus lower contractile speed (Barany 1967). They also possess the MHC isoform designated MHC-I (Staron 1997; McComas 1996; Bottinelli & Reggiani 2000). These fibres rely heavily on aerobic/oxidative energy metabolism and are classified as slow-twitch oxidative based on their aerobic/oxidative enzymatic activity (McComas 1996). Type-IIA fibres are fast-twitch muscle fibres based on myosin ATPase staining, possess MHC-IIA,

and are classified as fast-twitch oxidative since they often rely primarily on aerobic/oxidative metabolism (Staron 1997; McComas 1996; Bottinelli & Reggiani 2000). Type-IIX fibres (previously known as IIB) are fast-twitch muscle fibres, but they possess the MHC-IIX isoform (Bottinelli & Reggiani 2000) and are classified as fast-twitch glycolytic since they rely primarily on anaerobic/glycolytic energy metabolism (McComas 1996; Bottinelli & Reggiani 2000). It is important to note that human muscle fibres are now considered *not* to possess the MHC-IIB isoform found in other mammalian species (Bottinelli & Reggiani 2000), however much past, as well as current literature, still refers erroneously to human type-IIX fibres as type-IIB (e.g. Vollestad & Blom 1985). Within the muscle, these fibre types are organized into motor units, consisting of a motor neuron and the muscle fibres that it innervates. All muscle fibres within a given motor unit consist of only one fibre type. Therefore, motor-units are often referred to as type-I, type-IIA, or type-IIX motor units, or as lower and higher-threshold motor units, respectively.

Children's lower maximal isometric strength, lower short-term power, and slower force kinetics relative to adults can be explained by lesser type-II motor-unit activation. Type-II fibres are larger in diameter and thus generate more force when recruited (Bottinelli & Reggiani 2000). Importantly, they generate more force per unit cross-sectional area (Bottinelli & Reggiani 2000). They also contract much faster than type-I motor units owing to their higher myosin ATPase activity (Herbison et al. 1982; Bottinelli & Reggiani 2000). Children's faster recovery from high-intensity, short-term exercise can be tied to this lower power output stemming from lesser type-II motor-unit use and the resultant lower lactate response to exercise, resulting in children having less

to recover from (Falk & Dotan 2006). Lower type-II motor-unit activation can also explain children's enhanced muscular endurance given that children would utilize their type-I motor-units to a greater extent during muscular performance. Type-I muscle fibres show greater endurance and lower fatigue rate during sustained or repetitive contractions, as well as lower lactate production than type-II fibres due to their greater efficiency in energy metabolism and higher oxidative capacity (Herbison et al. 1982).

Lesser type-II motor-unit activation in children can account for the observed child–adult differences in metabolic responses to exercise since reduced type-II motor-unit use would elicit a much lower production of lactate in the muscle and subsequent accumulation in the blood (Falk & Dotan 2006). Type-IIX motor-units are much less efficient than type-I motor-units in terms of their energy metabolism and oxidative capacity, such that they rely almost exclusively on glycolytic metabolism (Herbison et al. 1982), breaking down glucose to generate lactate. Thus, children's lower activation of type-II motor-units would be consistent with their lower peak lactate response and higher lactate threshold during exercise. Faster phosphocreatine recovery following intense exercise, such as that observed in children relative to adults, is also thought to indicate a more oxidative, less glycolytic muscle metabolic profile with an associated greater reliance on oxidative muscle fibre types (Arnold et al. 1984). Lower type-II motor-unit activation in children, and consequently a higher reliance on type-I motor-units, can further explain children's higher rates of fat oxidation compared with adults (Riddell et al. 2008). Beta-oxidation of fatty acids produces a very high ATP yield, thus the increased fat metabolism of type-I muscle fibres contributes greatly to their energy efficiency and endurance, reducing their reliance on carbohydrate metabolism for the energy required to

continue contractions. Children's apparently faster phase-II pulmonary VO_2 kinetics can also be explained by lesser type-II motor-unit recruitment given that those with higher functional type-I muscle-fibre composition and corresponding higher muscle oxidative capacity would be expected to show such a VO_2 response (Barstow et al. 1996).

The lower neuromuscular activation in children relative to adults, described above, could reflect a lower total motor-unit recruitment, rather than specifically type-II motor-unit use. However, based on the progressive glycogen depletion patterns in muscle fibres during prolonged (Vollestad et al. 1984), or progressive exercise (Vollestad & Blom 1985), the lower threshold type-I motor units are believed to be recruited first. Thus, the lower overall neuromuscular activation observed in children could reasonably indicate lower activation of the larger, higher threshold type-II motor-units, which are activated later (Henneman et al. 1965; Vollestad et al. 1984; Vollestad & Blom 1985). While it is possible that recruitment order may also differ between children and adults, evidence in support of this suggestion is currently unavailable.

Children's lower rate of muscle activation, as reflected by their lower Q_{30} index values, may also be explained by lower type-II motor-unit activation, given that Q_{30} has been suggested to be related to movement velocity and to increase with training (Gabriel & Boucher 2000; Cohen et al. 2010; Mitchell et al. 2011). For example, high Q_{30} values were observed in young male gymnasts, whose training typically consists of a large number of explosive muscular contractions. On the other hand, young male endurance-trained swimmers with similar peak torque, had lower peak rates of torque development and Q_{30} values, which were similar to those of the untrained controls (Mitchell et al. 2011). Finally, the child–adult differences in mean power frequency might also reflect

children's lower type-II motor-unit activation since MPF has been shown to be affected by fibre-type composition (Komi & Tesch 1979), to increase following explosive training (Gabriel et al. 2001), and has been suggested to decrease during fatiguing exercise (*i.e.* shift leftward towards lower frequencies) due to the faster fatigue of recruited type-II motor-units (Kamen & Gabriel 2010; Gabriel et al. 2001; Halin et al. 2003; Armatas et al. 2010).

All of the differences examined above can be explained by children's lower activation of type-II motor units. However, these differences might also be explained by child–adult differences in muscle composition.

Nevertheless, the existing body of supporting evidence for the differential motor-unit activation hypothesis is extensive. However, the evidence obtained thus far has been indirect. In actual fact, until the appropriate techniques are developed to allow for large scale motor-unit activation monitoring in relation to force development and timing patterns, this hypothesis will continue to rely on such indirect evidence (Dotan et al. 2012).

Limitations of the EMG signal

Electromyography (EMG) is an evaluative tool that allows for the monitoring of skeletal muscle electrical activity. A number of variables can be derived from the EMG signal, such as the rate of EMG rise (Q_{30}), electromechanical delay, and mean power frequency. However, two main limitations are inherent to EMG signal collection that present a major issue in trying to compare MU activity between individuals, and more importantly between children and adults. The first limitation is cross-talk, whereby the recorded EMG signal may pick up motor-unit action potentials from nearby muscles

other than the muscle of interest (Gerdle et al. 1999). This issue is likely to be more significant when recording in the child group given their smaller muscle size. The surface EMG electrode head is the same size for both the adult and child groups, such that in the children there will be more muscles within the recorded area of the electrode site that could contribute to cross-talk. Cross-talk would alter the duration and amplitude of recorded EMG bursts, as well as increase "noise" in the baseline activity between successive EMG bursts.

The second, and most important, limitation is that direct comparison of EMG signal amplitude cannot be made between individuals, nor even within the same individual between separate testing sessions. In particular, the differences in EMG signal characteristics between children and adults would be expected to be vary greatly since EMG timing and duration is known to vary with age and body size, and the within session variation in the EMG signal has been shown to be far greater in children than adults (Benedetti et al. 2010).

The Electromyographic Threshold (EMG_{Th})

The electromyographic threshold (EMG_{Th}) is another variable derived from the EMG signal and represents a potential source of evidence that could help in substantiating the differential motor-unit activation hypothesis since it may provide the best method to date of distinguishing between type-I and II motor-unit involvement during exercise. The EMG threshold (EMG_{Th}) is defined as a non-linear increase or upward inflexion in the EMG signal during progressively increasing exercise intensity. This sharp increase in EMG activity at the EMG_{Th} intensity has been proposed to reflect

the onset of increased recruitment of type-II motor-units needed to maintain the energy supply and force required to continue contractions as muscle fibres recruited earlier begin to fatigue (Edwards & Lippold 1956; Petrofsky 1979; Nagata et al. 1981). This view that the EMG_{Th} reflects the onset of increased type-II motor-unit recruitment has since been widely accepted (Green & Patla 1992; Moritani et al. 1984; Taylor & Bronks 1994; Maestu et al. 2006; Hug et al. 2003, 2006a; Chwalbinska-Moneta et al. 1994, 1998; Bearden & Moffatt 2001; Takaishi et al. 1992; Hug & Dorel 2009). Figure 1 below shows an example illustrating the EMG_{Th}.

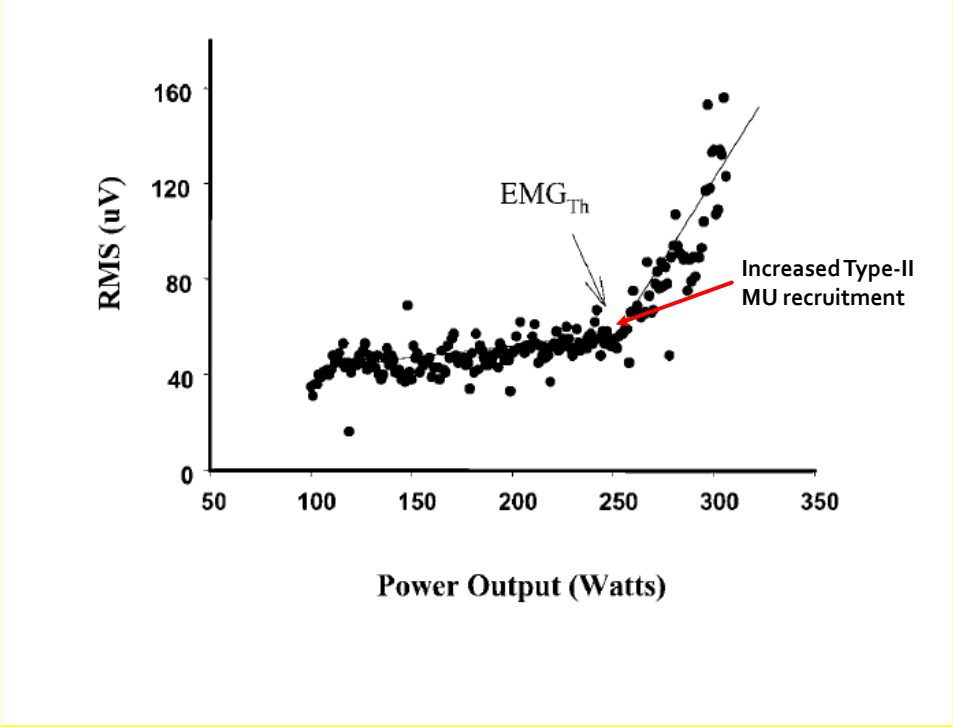

Figure 1 Example of **EMG_{Th}** in a male adult Rectus femoris muscle, elicited via progressive cycling exercise. From Hug et al. 2006.

The major advantage of examining the EMG_{Th} in comparing differential MU activity between children and adults is that there is no need for direct EMG signal comparison between participants since EMG_{Th} is expressed as a percentage of peak

exercise intensity which can be compared on the same scale between individual participants and between participant groups.

It might be argued that the large increase in EMG activity is simply a reflection of increased total motor-unit recruitment and/or firing frequency as the exercise intensity increases, rather than specifically type-II motor-units. However, in accordance with the size principle (Henneman et al. 1965), orderly recruitment of muscle fibres beginning with type-I fibres and ending with type-IIX fibres would reasonably be expected. Vollestad & Blom (1985) studied glycogen depletion of type-I, IIA, IIAB (currently referred to as IIA-IIX), and IIB (currently referred to as IIX) muscle fibres during cycling at 43%, 61%, and 91% of VO_{2max} in highly fit men (mean VO_{2max} 59.6±2.5 $ml \cdot kg^{-1} \cdot min^{-1}$, range 53–67 $ml \cdot kg^{-1} \cdot min^{-1}$). At 43% VO_{2max}, glycogen depletion occurred in all type-I and 20% of type-IIA fibres (Figure 2). At 61% VO_{2max}, glycogen was depleted from all type-I and 65% of type-IIA fibres. At 91% VO_{2max}, glycogen depletion was observed in all type-I and type-IIA fibres, while type-IIAB (IIA-IIX) and type-IIB (IIX) fibres showed about 50% depletion, indicating a later recruitment of these more glycolytic fibre types during the progressive exercise test.

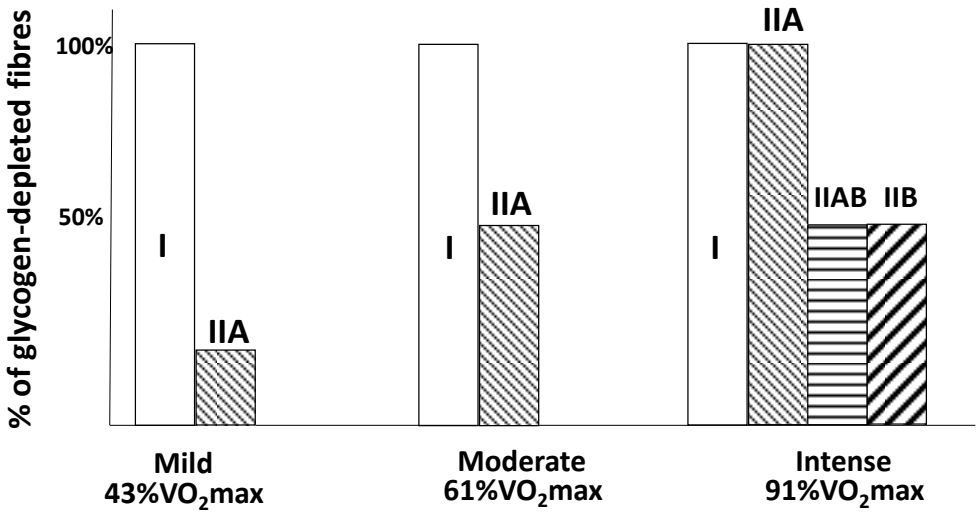

Figure 2. Glycogen depletion in type-I, IIA, IIAB, and IIB muscle fibres during progressive cycling exercise in men. Based on Vollestad & Blom 1985

In a different study, Vollestad et al. (1984) examined glycogen depletion in the same four fibre types during different durations of prolonged exercise at a moderately high intensity (75% VO_{2max}), corroborating the findings from the progressive exercise tests. They observed that the rates of glycogen depletion were equivalent for the oxidative type-I and IIA fibre types from the onset of exercise, indicating early recruitment of these fibre types, while glycogen content of type-IIAB (IIA-IIX) and IIB (IIX) fibres did not change. As exercise continued, glycogen was first depleted from type-IIAB (IIA-IIX) fibres, then finally from type-IIB (IIX) fibres. These results suggest a progressive recruitment of fibre types during both graded and steady-state exercise tests, beginning with the lower

threshold, oxidative type-I and IIA motor-units and ending with the higher threshold, glycolytic type-IIAB (IIA-IIX) and IIB (IIX) motor-units.

It should be noted that since Vollestad & Blom (1985) and Vollestad et al. (1984) used cycle-ergometry, what they refer to as VO_2max is normally defined as VO_2pk (as in the present study).

That the EMG_{Th} reflects the onset of increased type-II motor-unit recruitment is further supported by studies of muscle fibre conduction velocity. Muscle fibre conduction velocity relates to the contractile and sarcolemmal membrane properties of muscle fibres, with the lower threshold, typically smaller diameter type-I fibres showing slower muscle fibre conduction velocity than the higher threshold, larger diameter type-II fibres (Farina et al. 2004a, b). Accordingly, during progressive cycle ergometer exercise, Farina et al (2004a) found increased muscle fibre conduction velocity (MFCV) at higher power outputs in healthy men (age: 26±5yrs; 4 of 12 were trained cyclists). Subjects cycled at different cadences and at power outputs that were equivalent to 50% and 100% of their lactate threshold (Farina et al. 2004a). At each cadence, MFCV was higher at the higher power output. The authors concluded that the increasing MFCV at progressively higher power outputs "indicate[d] progressive recruitment of large, high conduction velocity motor units with increasing muscle force" (Farina et al. 2004a).

Given that the EMG_{Th} is generally viewed as reflecting the onset point of accelerated type-II motor-unit recruitment, finding child–adult EMG_{Th} differences may be the best source of indirect evidence currently available that would support the differential motor-unit activation hypothesis.

Factors affecting the EMG threshold

A number of different factors could potentially affect detection of an EMG_{Th} or the point of EMG_{Th} occurrence, including exercise mode and protocol, training status, muscle examined, and age.

Exercise mode: Numerous studies have demonstrated the EMG_{Th} phenomenon during various exercise modes. For example, an EMG_{Th} has been observed during incremental forearm exercise (Moritani et al, 1984), treadmill running, (Taylor & Bronks 1994; Tikkanen et al. 2012), ergometer rowing (Maestu et al. 2006), and, most notably cycle ergometer exercise tests (Hug et al. 2003, 2004a, 2006a; Chwalbinska-Moneta et al. 1994, 1998; Bearden & Moffatt 2001; Lucia et al. 1999; Moritani et al. 1993; Takaishi et al. 1992; Nagata et al. 1981; Viitasalo et al. 1985).

Varied involvement of different muscle groups could possibly produce different patterns of motor-unit recruitment between exercise modes. Such differential neuromuscular activation among different exercise modes could alter EMG_{Th} determination. For example, during treadmill running, the unrestricted stride length and frequency, relatively higher muscle mass recruited (compared with cycling), and inefficient running technique of inexperienced runners may all contribute to variations in muscle activation patterns and thus also to variation in the resultant EMG signal from a given muscle (Tikkanen et al. 2012). In the case of inexperienced runners, poor muscle coordination could lead to a higher degree of co-contraction and possible activation of unnecessary muscles. The EMG_{Th} might thus be expected to occur earlier since inexperienced runners would "elicit a higher EMG value than what is needed to achieve a given running speed" (Tikkanen et al. 2012).

During cycling, factors thought to influence EMG patterns include shoe-pedal interface, body position, fatigue, pedaling rate, and training status (Hug & Dorel 2009).

Shoe-pedal interface: Shoe-pedal interface can alter recorded EMG activity due to differing pedaling mechanics with different pedal types (Hug & Dorel 2009). For example, Ericson (1986) compared EMG activity level using standard platform pedals and toe-clip pedals used with cycling shoes. Using toe-clip pedals, higher EMG activity was observed in the *tibialis anterior, rectus femoris,* and *biceps femoris* muscles, while lower EMG activity was found in the *soleus, vastus medialis* and *vastus lateralis* muscles (Ericson 1986). While the effects of shoe-pedal interface on the EMG_{Th} occurrence are unknown, any systematic effect can be minimized by using the same shoe and pedal type for all participants.

Body Position: Variables of body position, such as seat height, and trunk and hip angles, can modify muscle activation patterns (Hug & Dorel 2009). For example, Jorge and Hull (1986) found quadriceps and hamstrings activity to be higher at a seat height of 95% of trochanter length compared to a seat height of 100% of trochanter length, with any further increases in seat height (keeping handlebar height constant) leading to decreases in the quadriceps activity level. Decreasing the trunk to hip angle (*i.e.* crouched forward cycling position, keeping seat height constant) increases activation of the *gluteus maximus* (Savelberg et al. 2003), while moving from seated to standing position also increases activity of the *gluteus maximus, rectus femoris, tibialis anterior*, and *vastus lateralis* muscles (Li & Caldwell 1998). This differential EMG activity for specific muscles with varying body positions could affect the exercise intensity at which the EMG_{Th} would occur. For example, if recording EMG activity from the vastus lateralis,

participants moving from a seated to a standing position would have higher EMG activity for any given power output. This would likely elicit an earlier EMG_{Th} compared with participants maintaining a seated position. Thus, to minimize these effects of body position, a standard seat height and body position, based on leg length and trunk height, should be adopted for all participants.

Fatigue: Fatigue would be manifested by rising EMG activity with exercise progression towards volitional exhaustion. This is thought to occur due to progressive recruitment of additional motor units needed to make up for those units that have fatigued, as described previously (Vollestad & Blom 1985; Vollestad et al.1984). However, changing EMG activity patterns during fatiguing exercise could potentially also be explained by changing muscle coordination patterns, altered muscle contractile properties, changes in the timing of muscle activation, and altered muscle fibre conduction velocity (see Hug & Dorel 2009 for review). Some studies have nevertheless demonstrated a clear EMG_{Th} using intermittent exercise protocols with recovery intervals to minimize these effects of fatigue (*e.g.*, Chwalbinska-Moneta 1994, 1998).

Cadence: Conflicting results exist in the literature as to the exact relationship between pedaling rate and EMG activity in the individual muscles involved in cycling (see Hug & Dorel 2009 for review). Some studies report increased EMG activity in certain muscles as cadence increases, with no change in other muscles (Ericson 1986; Neptune et al. 1997; Sarre et al. 2003), while other studies report decreased EMG activity with increasing cadence (Lucía et al. 2004). These discrepant results are likely best explained by differences in the power outputs, range of cadences, and the training status of subjects tested, as well as the distinct response to pedaling rate changes of different

individual muscles (Hug & Dorel 2009). The use of different types of cycle-ergometers may also account for some of the discrepancy. For example, in using iso-power ergometers, increasing pedaling cadence inversely affects the resistance and reduces the applied muscle force with each pedal stroke, which would be expected to directly affect the motor-unit recruitment pattern. Studies have shown that as pedaling rate increases, peak EMG amplitudes in various lower-limb muscles involved in cycling occur earlier in the pedaling cycle (Marsh & Martin 1995; Neptune et al. 1997). Similarly, EMG activity varies with cadence at any given power output, with the minimum EMG activity level occurring at progressively higher cadences as power output is increased (MacIntosh et al. 2000). Thus, at higher power outputs, maintain a slower cadence requires more applied force and will elicit higher EMG activity than a faster cadence. This would affect EMG_{Th} occurrence since at slower cadences (*e.g.*, 60 rpm), EMG activity would increase more steeply with increasing power output compared with a faster cadence (*e.g.*, 90 rpm). It is therefore important to keep the pedalling rate constant for all participants, as well as within a given test, in order to minimize this effect.

Exercise protocol: Within a given exercise mode, the specific exercise protocol could also affect muscle activity patterns and EMG_{Th} determination. Incremental exercise protocols consist of increasing the power output by a set step increment, either in discrete stages of specific duration, or in continuous, ramped fashion. Different protocols have been utilized in the various studies investigating the EMG_{Th}, with similar results. For example, both Hug et al. (2003) and Lucia et al. (1999) found EMG_{Th} s to occur at corresponding exercise intensities, in the same muscles, in subjects of comparable training status, despite employing different protocols (ramp of 26W per minute and 5W

every 12 seconds, respectively). Takaishi et al. (1992) examined the EMG_{Th} phenomenon using iEMG recorded from the vastus lateralis during four different ramp slope increments (10, 20, 30, and 40 watts per minute). The VO_2 at the iEMG breakpoint (*i.e.* EMG_{Th}) showed no significant difference between the four ramp increments. That is, the EMG_{Th} occurred at the same percentage of VO_2pk regardless of the ramp slope increase per stage (Takaishi et al. 1992). These findings appear to suggest that during an incremental exercise protocol the EMG_{Th} can be determined and that slight variations in the protocol should not affect the intensity at which the EMG_{Th} is observed. However, based on extensive pilot testing, a ramped protocol appears to facilitate EMG_{Th} identification, compared with a protocol of stepwise increments.

Training status: Subjects' training status has the potential to modify EMG characteristics and muscle recruitment patterns based on neuromuscular adaptations acquired as a result of repeated task performance (Osu et al. 2002; Schneider et al. 1989). Particularly in cycling, trained cyclists show increased EMG activity and hypertrophy of m. *biceps femoris* compared with non-cyclists, suggesting neuromuscular adaptation associated with enhanced pedaling skill (Takaishi et al. 1998; Hug et al. 2004b). Chapman et al. (2008) found further differences in recruitment of five lower limb muscles between highly trained and novice cyclists. Compared with the former, novice cyclists showed greater individual and population variance in EMG amplitude and duration, more variable muscle co-activation, and greater EMG amplitude in periods between primary EMG bursts, suggesting different muscle recruitment strategies between the two groups (Chapman et al. 2008). It is likely that these differences in muscle recruitment between trained and untrained cyclists could have a marked effect on EMG_{Th}

occurrence. Highly trained endurance athletes would be expected to utilize type-I and aerobically trained type-IIA motor-units more heavily during progressive exercise. For such endurance athletes, a large rise in type-II motor-unit recruitment would be expected to occur only at the very end of the exercise, at a higher relative exercise intensity than non-athletes. That is, the EMG_{Th} would occur later for highly trained endurance athletes.

Another possible training-dependent difference is the manifestation of multiple EMG thresholds. Studies of non-cyclists typically demonstrate a single EMG_{Th} (Hug et al. 2004a, 2006a; Chwalbinska-Moneta et al. 1994, 1998; Moritani et al. 1993; Takaishi et al. 1992; Nagata et al. 1981; Viitasalo et al. 1985), but in professional road cyclists Hug et al. (2003) found two EMG_{Th} s in eight lower limb muscles, at mean intensities of 52% and 86% of maximal power output. Lucia et al. (1999) also observed two EMG thresholds in m. vastus lateralis and rectus femoris of elite road cyclists, which occurred between 60-70% and 80-90% VO_2max. Bearden & Moffatt (2001) found two EMG breakpoints at 58% and 75% of VO_2pk in m. *vastus lateralis* of trained male cyclists. It seems likely that fast-twitch, oxidative, type-IIA (and possibly, the intermediate type-IIA-IIX) fibres are recruited at the first EMG breakpoint, while the second EMG breakpoint indicates the increased recruitment of the glycolytic type-IIX (and possibly additional IIA-IIX) fibres (Tikkanen et al. 2012).

The twin threshold phenomenon observed in cycling does not appear to persist in other exercise modes. In rowing-ergometer tests to exhaustion, national-level competitive rowers showed only a single EMG_{Th} (Maestu 2006). The same was observed for national-level endurance-trained runners during treadmill running tests (Tikkanen et al. 2012). Moreover, any training effect may reflect task-specific or sport-specific training, rather

than an effect of overall high fitness, given that only a single EMG_{Th} was determined in speed-trained sprinters and endurance-trained cross-country skiers during incremental cycling exercise (Chwalbinska-Moneta et al. 1994).

Muscle examined: The EMG_{Th} has been observed in various muscles. In cycling, EMG_{Th}s have been detected for many of the hip, knee, and ankle flexor and extensor muscles utilized during pedaling. An EMG_{Th} has been observed for the *vastus lateralis* (Hug et al. 2003, 2006a; Nagata et al. 1981; Lucia et al. 1999; Takaishi et al. 1992; Bearden & Moffatt 2001), *rectus femoris* (Chwalbinska-Moneta et al. 1994,1998; Hug et al. 2003, 2006a; Lucia et al. 1999), *soleus* (Chwalbinska-Moneta et al. 1994,1998), *biceps femoris* and *gastrocnemius lateralis* (Bearden & Moffatt 2001; Hug et al. 2003, 2006a), as well as the v*astus medialis, gastrocnemius medialis, semimembranosus,* and *tibialis anterior* muscles (Hug et al. 2003, 2006a). It could be expected that muscles higher in type-I MUs would show a relatively later EMG_{Th} than muscles with predominantly type-II MUs, depending of course upon the extent to which a given muscle is used during the particular activity. For example, EMG_{Th} was shown to occur earlier in the type-I fibre-rich *soleus* muscle compared with type-II fibre-rich *rectus femoris* (Chwalbinska-Moneta et al. 1994), suggesting a more complicated relationship between muscle fibre composition and the point of EMG_{Th} occurrence. Along those lines, the point of EMG_{Th} occurrence (as percentage of peak power output) did not differ between several different lower limb muscles of varying muscle fiber composition (Hug et al. 2006a), suggesting systemic factors might also influence the EMG_{Th} in addition to local mechanisms at the level of the individual muscle. However, the reliability and repeatability of EMG_{Th} occurrence has been found to vary depending on the muscle

examined (Hug et al. 2003, 2006a; Dorel et al. 2008; Laplaud et al. 2006). For example, Hug et al. (2003) could detect a threshold in only 50% of *gastrocnemius lateralis* muscles tested, compared with a 100% detection rate in the *vastus lateralis*. The latter showed 100% test-retest reliability, while EMG_{Th} occurrence in several other lower limb muscles (especially the *rectus femoris, gastrocnemius medialis,* and *semimembranosus*) was much less reliable (Hug et al. 2006a). These findings may be expected given that *vastus lateralis* is the principal muscle involved in pedaling, while the other lower limb muscles can be used more variably (Hug & Dorel 2009).

This variability in EMG_{Th} occurrence between muscles may be explained by differential muscle activity levels and timing of activation patterns in cycling (Hug & Dorel 2009). For example, Ericson (1986) found higher EMG activity levels in the mono-articular muscles (*e.g. vastus lateralis* and *medialis, soleus*) compared with bi-articular muscles (*e.g., rectus femoris, gastrocnemius lateralis*). Laplaud et al. (2006) further found poor inter-session reproducibility in terms of muscle activity level and activation timing in the *rectus femoris*, while Dorel et al. (2008) had similar findings of poor reproducibility for the *rectus femoris*, *soleus*, and *tibialis anterior* muscles. This high variability across individual muscles and between subjects suggests variable individual muscle activation patterns (Laplaud et al. 2006). However, it seems likely that the factors of influence described above may also affect the EMG characteristics of individual muscles. For example, training status may play a role. Along with the *vastus lateralis*, Hug et al. (2003) found 100% EMG_{Th} occurrence in the *biceps femoris* muscle of professional road cyclists, compared with a much lower rate of detection in sedentary subjects (Hug et al. 2006a). It may be that the reliability of EMG_{Th} occurrence in the

biceps femoris of trained cyclists reflects the increased EMG activity and hypertrophy of the *biceps femoris* associated with enhanced pedaling skill (Takaishi et al. 1998; Hug et al. 2004b).

Apart from the lower limb muscles involved in cycling, Moritani et al. (1984) found an EMG_{Th} in the forearm *flexor carpi radialis-palmaris longus* during incremental forearm exercise to exhaustion, suggesting the EMG_{Th} may be a universal skeletal muscle phenomenon. Similarly, Chwalbinska-Moneta et al. (1994) found an EMG_{Th} in the nonworking *frontalis* muscle of the forehead during progressive cycling exercise to exhaustion. This finding suggests a central nervous system component could be involved in increasing whole body motor unit activation, either separately or in conjunction with local fatigue mechanisms that would elicit increased motor-unit recruitment within the working muscles.

Age: While the EMG_{Th} has been observed under numerous experimental conditions for a variety of exercise modes in adults, to our knowledge it has not yet been examined in children. If children, as suggested earlier, do indeed have a different type-II motor-unit recruitment pattern compared with adults, then this difference would be expected to manifest itself as a differential EMG_{Th} as well. Along these lines, children have been shown to have lower muscle activation during maximal voluntary isometric contractions in the *quadriceps* and *triceps surae* muscle groups (Stackhouse et al. 2005, O'Brien et al. 2009). Similar age-related differences could be expected for tasks involving dynamic contractions, such as cycling. However, research into this area in children is limited.

Kaplan (1995) found EMG evidence of considerably greater *tibialis anterior* and lateral *gastrocnemius* muscle co-contraction during cycling in 10-year-old children compared with adults. However, co-contraction levels of the *rectus femoris* and medial hamstring muscles were comparable with typical adult values. This difference might be attributable to differences in inter-muscular coordination and joint power contributions to total pedal power between children and adults during the cycling task (Korff et al. 2009).

Korff et al. (2009) found that compared with adults, children produced a significantly smaller proportion of total pedal power at the ankle joint (only about half as much as adults) during a 3-second maximal-power cycling trial. Children compensated by generating relatively more power at the knee and hip joints (Korff et al. 2009). While the increased co-contraction of the ankle muscles might have an effect on children's pedaling mechanics, this effect is likely minimal given the generally low involvement of the ankle joint in power generation. EMG recordings from the thigh muscles (*i.e.* hamstrings, *quadriceps*, and in particular the *vastus lateralis*) should thus be largely unaffected by this child–adult difference in ankle involvement. However, in light of the lack of data concerning children's cycling EMG characteristics, it is hard to predict how EMG activity levels, timing of muscle activation, and ultimately EMG_{Th} occurrence might differ between children and adults.

Relationship between the EMG- and metabolism-related thresholds

The EMG_{Th} has been associated with various metabolic, aerobic-anaerobic transition points, such as the lactate threshold, the first and second ventilatory thresholds, and the onset of blood lactate accumulation (OBLA) (refer to Appendix A, Table 5 for summary). It is important to note that these metabolic thresholds do not necessarily occur

at the same point since they do not necessarily represent the same physiological processes. Moreover, while the EMG_{Th} reflects local, neuro-motor changes, the OBLA, lactate, and ventilatory thresholds are systemic, reflecting metabolic changes in other, differently-active muscles, as well.

Studies have shown significant correlations between the EMG_{Th} and the blood lactate threshold (Moritani et al. 1984; Chwalbinska-Moneta et al. 1998; Moritani et al. 1993; Nagata et al. 1981), OBLA (Tikkanen et al. 2012), and the ventilatory threshold (Nagata et al. 1981; Tikkanen et al. 2012).

A number of studies have also found the EMG_{Th} to occur at similar exercise intensities as the lactate threshold (Chwalbinska-Moneta et al. 1994, 1998; Lucia et al. 1999), the ventilatory threshold (Hug et al. 2003; Lucia et al. 1999; Maestu et al. 2006; Tikkanen et al. 2012), and OBLA (Lucia et al. 1999; Tikkanen et al. 2012).

Some inconsistency in the literature does, however, exist. Taylor & Bronks (1994) found the EMG_{Th} to occur at a significantly greater exercise intensity than the lactate threshold (79.8 *vs.* 72.2% VO_2pk, respectively). However, this finding might be expected since the lactate threshold is very variable and occurs earlier than the anaerobic threshold. Therefore the EMG_{Th} might reasonably be found higher than the lactate threshold. This would suggest that lactate accumulation is not the only factor attributable to the motor unit recruitment changes occurring at the EMG_{Th} (Taylor & Bronks 1994) and is more likely a reflection of changing motor-unit recruitment patterns during exercise. Hug et al. (2003), on the other hand, found in trained cyclists showing two thresholds that the first EMG_{Th} occurred significantly before the first ventilatory threshold, at 52% and 62% of maximum power output, respectively. The authors explain this discrepancy by

highlighting that the ventilatory threshold reflects all the muscles involved in the exercise while the EMG_{Th} is determined separately for individual muscles (Hug et al. 2003).

Regardless of these divergent results, based on the overall findings, there seems to be a clear association between the EMG_{Th} and the other metabolic thresholds indicative of a shift towards anaerobic metabolism. It does indeed seem reasonable that an increased activation of the more fatigable, less oxidative type-II motor units thought to occur at the EMG_{Th} should lead to higher lactate production in working muscles, higher lactate accumulation in the blood, and produce the additional metabolic and ventilatory responses that are reflected by the ventilatory threshold and OBLA.

Given that the EMG_{Th} is thought to reflect the onset point of increased type-II motor-unit recruitment, examining the EMG_{Th} in children provides the nearest measurement to date of type-I and II motor-unit involvement during exercise and should prove very valuable in providing support for the differential motor-unit activation hypothesis.

Objective

The objective of this study was to compare the relative exercise intensity at which the EMG_{Th} occurs in boys and men. Male participants were chosen given that the child–adult differences between boys and men are often more apparent than between girls and women (*e.g.*, Falk et al. 2009a, b).

Hypothesis

Given the close correlation between the EMG_{Th} and the lactate and ventilatory thresholds described above, as well as the observation that the latter thresholds occur at a

higher relative exercise intensity in children relative to adults, it was hypothesized that the EMG_{Th} would also occur at a higher relative exercise intensity in boys compared with men. Such findings would reflect a later onset of increased recruitment of type-II motor-units in children and provide support for the differential motor-unit activation hypothesis.

Anticipated Significance

If shown to be true, children's higher EMG_{Th} could provide the strongest evidence yet for the differential motor-unit activation hypothesis.

Ultimately, the results should prove informative in elucidating the factors responsible for the numerous functional and physiological child–adult differences in muscular performance, neuromuscular function, and metabolic responses to exercise.

Methods

Design: The study's design was cross-sectional. The relative exercise intensity at which the EMG_{Th} occurred was compared between boys and men to indirectly assess how neuromuscular activation differs between the two groups.

Participants: Two groups of participants were included: boys (n=23; 11.1±1.1 yrs) and men (n=21; 23.4±4.1 yrs) (Table 1). Participants were excluded if they demonstrated any of the following criteria:

1. Any risk factors and past or present muscular disease.
2. Chronic/frequent use during the preceding year, of medications that could affect neuromuscular function.
3. Injury to either leg, or any injury that could impede cycling performance.

Study Procedures: All tests and measurements were performed during two visits to the Applied Physiology Laboratory, Department of Kinesiology, at Brock University. The study was reviewed and received ethics clearance though the Brock University Research Ethics Board (file #12-209). During Visit 1, participants were informed of all tests and procedures to take place over both visits. Anthropometric measurements (height, seated height, mass, percent body fat using skinfold calipers, limb length and circumference) were taken. Questionnaires regarding any medical concerns, physical activity habits, training history, and pubertal stage (Tanner, 1962) were completed (see Appendices). Finally, seat and handlebar heights, positioning, and crank length on the cycle-ergometer were determined and adjusted per the participant's body size. Participants performed a progressive cycling test to volitional exhaustion, while expiratory gases were collected to determine VO_{2max}. During Visit 2, a measurement of the vastus lateralis cross-sectional area (muscle depth) was performed using ultrasound (See Appendix B), along with identification of the origin and insertion points. These measurements were used to estimate muscle volume. After preparing the skin, attaching the electrodes, and setting the same cycle-ergometer seat, handlebar, and crank length settings, participants performed the EMG_{Th} test (see protocols below).

Measurements

Body stature: Height was measured to the nearest 0.1cm using a stadiometer (Ellard Instrumentation Ltd.).

Mass: Total body mass was measured to the nearest 0.1kg using a digital scale (InBody 520, Biospace CO., Ltd). Participants removed their shoes and any excessive clothing that may significantly affect their weight.

Skinfold Thickness: Skinfold thickness was measured in triplicate using Harpenden calipers (British Indicators, Herts, England) and the median value at each site was be used. Skinfold thickness over the triceps and subscapular sites was be measured in order to estimate adiposity (percentage of body fat) using age- and maturity-specific equations (Slaughter et al. 1988). Skinfolds thickness of anterior, posterior, medial and lateral thigh were measured in order to evaluate thigh lean CSA (Gurney & Jelliffe, 1973). All measurements were performed by the same investigator, in order to eliminate inter-observer variability (Triceps + subscapular skinfolds Intraclass Correlation Coefficient (ICC) $_{(3, 1)}$=.99, *n*=14; thigh skinfolds $ICC_{(3, 1)}$=1.0, *n*=13)

Limb length and circumference: Circumference and length of the thigh were determined using methods as described in Lohman et al. (1988) to evaluate thigh lean CSA.

Muscle depth: Muscle depth was measured using a real-time B-mode ultrasound (System5, GE Vingmed, Horten, Norway) with 5 MHz linear-array probe. Longitudinal images of the vastus lateralis were obtained at rest. The scanning head of the probe was oriented along the mid-longitudinal axis of the anterior thigh, over a site at 61% the distance from the Anterior Superior Iliac Spine (ASIS) to the superior border of the patella. Muscle depth was measured as the distance between the more superficial adipose tissue-muscle interface and the deeper vastus lateralis–vastus intermedius interface, as previously described (Blazevich et al. 2006). All measures were performed by the same investigator in order to eliminate inter-observer variability (muscle depth $ICC_{(3, 1)}$=.99, *n*=11)

Cycle-ergometer settings: Prior to any testing on the cycle-ergometer, participants' preferred seat height/positioning, handlebar height/positioning, and crank length were adjusted and recorded. Crank length was determined based on inseam length according to the table from Smith et al. (1997) (see Appendix B).

Questionnaires

Medical History: Participants' medical history was determined using the Subject Screening Questionnaire (Appendix F) and their ability to complete the exercise component of the study assessed using the CSEP Physical Activity Readiness Questionnaire (PAR-Q & You).

Pubertal stage: The boys' pubertal status was self-assessed based on secondary sex characteristics (pubic hair) following the Tanner scale (Tanner 1962). Boys completed the Pubertal Stage Questionnaire (Appendix D) in a private space. The self-assessment form was placed in an envelope by the subject and handed directly to the researcher.

Leisure-time physical activity: Physical activity was determined using the questionnaire developed by Godin and Shephard (1985) (See Appendix E).

Training history: For participants who reported training in any sport, past and present training history was determined by a questionnaire and a personal interview (See Appendix G).

Exercise Protocols

Submaximal VO_2 and VO_2pk test (Visit 1): Participants assumed an upright position on the cycle-ergometer. Following a 5-minute warm-up period, participants

commenced an incremental exercise protocol on a cycle-ergometer (Excalibur Sport, Lode, Groningen, The Netherlands) consisting of 3–5 submaximal stages to determine the VO_2 *vs.* power relationship. The test progressed in 3.5-minute stages for the boys and 4-minute stages for the men, with increasing resistance (power output, in W) at each stage. Boys began between 40–60W and progressed in increments of 15–20W. Men began between 80-100W and progressed in increments of 30-40W. These submaximal stages were used to determine the VO_2 for several given power outputs and the determination of peak aerobic power (PO_2pk; the power output in Watts corresponding to VO_2pk) (see below).

Following the completion of 5 stages, participants were allowed a minimum of 15 minutes recovery. Participants then performed an incremental exercise protocol until volitional exhaustion to determine VO_2pk. Increments were increased every minute (10W/min and 20W/min, for the boys and men, respectively) until volitional exhaustion. Pedalling rate was maintained at a minimum of 80 rpm. Following VO_2pk determination, the resistance on the ergometer was reduced promptly and participants were allowed a cool-down period.

Heart rate was determined using a heart rate monitor (TIMEX Personal Heart Rate Monitor, TIMEX Group Inc., Toronto, ON) throughout the test. Ratings of Perceived Exertion were assessed at the end of each submaximal stage using the Borg RPE Scale (Appendix C). Expired gas was collected and analyzed using the Moxus metabolic cart (AEI technologies, PA). VO_2pk was determined in real-time as the average of the highest VO_2 values attained over three consecutive 15-second periods. This value was recorded in mL/kg/min.

Peak aerobic power (PO_2pk) determination: Data from the submaximal workloads and VO_2pk test were used to determine the power output (in Watts) corresponding to the absolute VO_2pk (ml/min), referred to as the PO_2pk. For each participant, a plot was constructed of VO_2 on the y-axis against Power Output on the x-axis. VO_2 values corresponding to each power output from the 5 submaximal stages were plotted and a linear regression line was fit to the data points. This line was extrapolated and the power output corresponding to the VO_2pk value was determined as the PO_2pk.

EMG Threshold test (Visit 2): Following a 5-minute warm-up period, participants performed a ramped incremental cycling test to exhaustion. Starting power output was set at ~35–40% of PO_2pk, and the ramp protocol was determined with the aim of having similar test durations for all participants (~10 min.). Thus, the mean starting power output for the boys was 51±12 W and power output increased on average 1W every 6 seconds, until volitional exhaustion. Mean starting power output for men was 103±21 W and power output increased on average 1W every 3 seconds, until volitional exhaustion. Surface EMG, using 10 mm^2, bipolar, Ag/Ag surface electrodes (Delsys 2.1, Delsys Inc., Boston, MA), was used to monitor the electrical activity of the *vastus lateralis* muscle of each leg continuously throughout the test. Participants were instructed to keep the pedalling rate as close to 80 rpm as possible throughout the test. Heart rate was monitored throughout the test using. Following volitional exhaustion, EMG recording was terminated and resistance on the cycle-ergometer was reduced promptly to allow participants a cool-down period.

Electrode placement: The *vastus lateralis* muscle was chosen as the muscle of interest given its reliability for EMG_{Th} determination as well as its high levels of

utilization and neuromuscular activity during cycling (Hug et al. 2003, 2006a; Laplaud et al. 2006; Dorel et al. 2008). Furthermore, the proportional changes in fibre type composition from higher type-I percentage to higher type-II percentage with age (Jansson 1996, Lexell et al. 1992), as well as progressive fibre-type recruitment from type-I to type-IIA/AB (IIA-IIX) to type-IIB (IIX) during progressive intensity exercise (Vollestad & Blom 1985) have been previously demonstrated in the *vastus lateralis* muscle (in men). An area of each thigh at two-thirds on the line from the anterior spina iliaca superior to the superior border of the patella was shaved (if necessary), abraded with skin prep gel, (Nuprep, Weaver and Company, Aurora, CO), and cleaned with rubbing alcohol. Surface electrodes were placed according to the Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) group recommendations for the *vastus lateralis* muscle (see Appendix H). Electrodes were placed parallel to the direction of muscle fibres on the medial aspect of the *vastus lateralis* and affixed with double-sided tape. An extra piece of single-sided 3M-Transpore adhesive tape was used to secure the electrode head in place, and another piece was used to tape the electrode cable to the thigh to avoid any movement artifacts during testing. A reference electrode was placed over the spinous process of the seventh cervical vertebrae and also secured in place with a piece of 3M-Transpore tape.

Technical Information: EMG signals were band-pass filtered (20–450 Hz) using the Bagnoli-4 (Delsys Inc., Boston, MA) bioamplifier and sampled at a rate of 1000Hz using a Computer-Based Oscillograph and Data Acquisition System (EMGworks Acquisition, Delsys Inc., Boston, MA). Recorded data were stored for further analysis.

EMG data reduction and analysis

EMG data were reduced and analyzed using a computer algorithm created in MATLAB v. 2013 (MathWorks Inc., Natick, MA) (Figure 3). With each pedal stroke, a succinct burst of EMG activity was produced and recorded from each leg. A raw waveform including consecutive right and left leg bursts was imported into MATLAB and pruned at the beginning and end to remove any partial or incomplete bursts. This pruned waveform was then de-trended to remove any deviation in the baseline (*i.e.* set the baseline value to zero) and filtered between 20–450 Hz using a high-pass Butterworth filter. The root mean square (RMS_{EMG}) was then calculated for the entire waveform. The onsets and offsets of each burst were then identified as the points where the EMG signal rose or fell above or below 10% of the mean RMS_{EMG} value for the

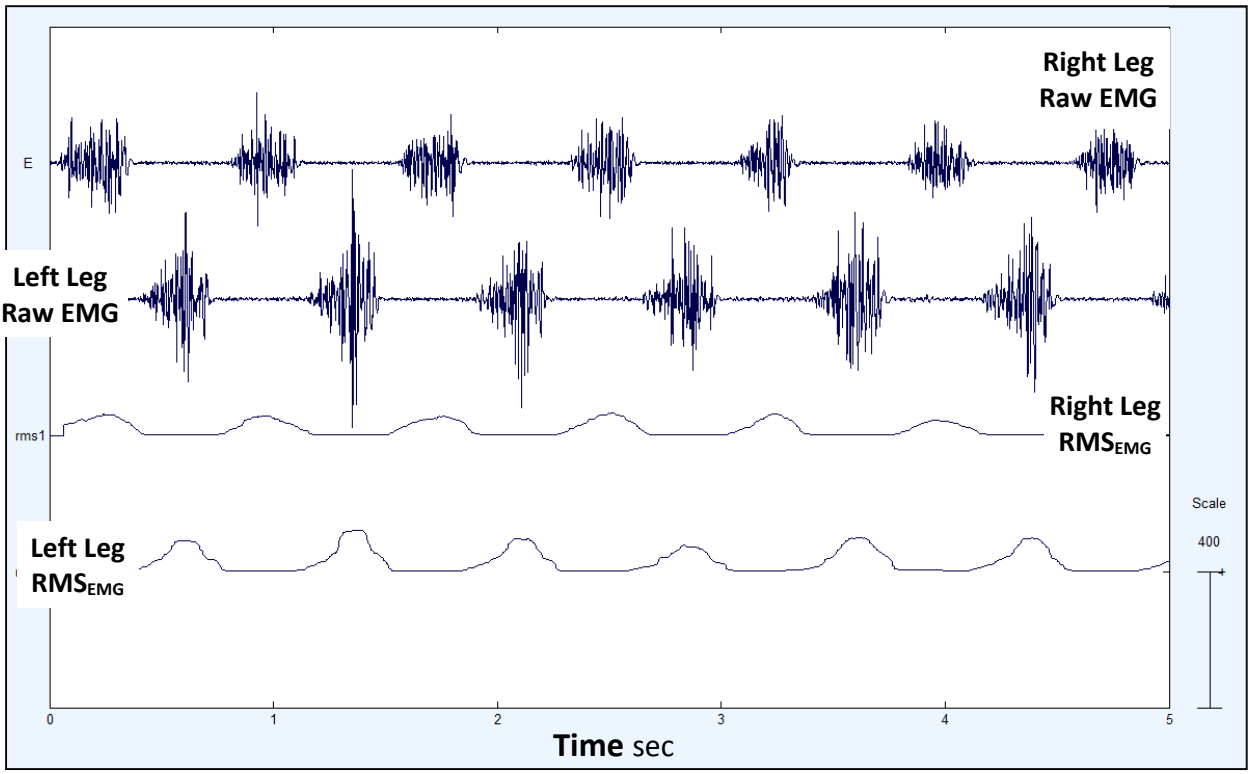

Figure 3. Example of a trace showing the raw EMG bursts and RMS_{EMG} for the right (top) and left (bottom) legs over 5 seconds.

entire test record (Figure 4). The RMS_{EMG} of each burst (*i.e.*, between the onset and offset) was then extracted for EMG_{Th} determination.

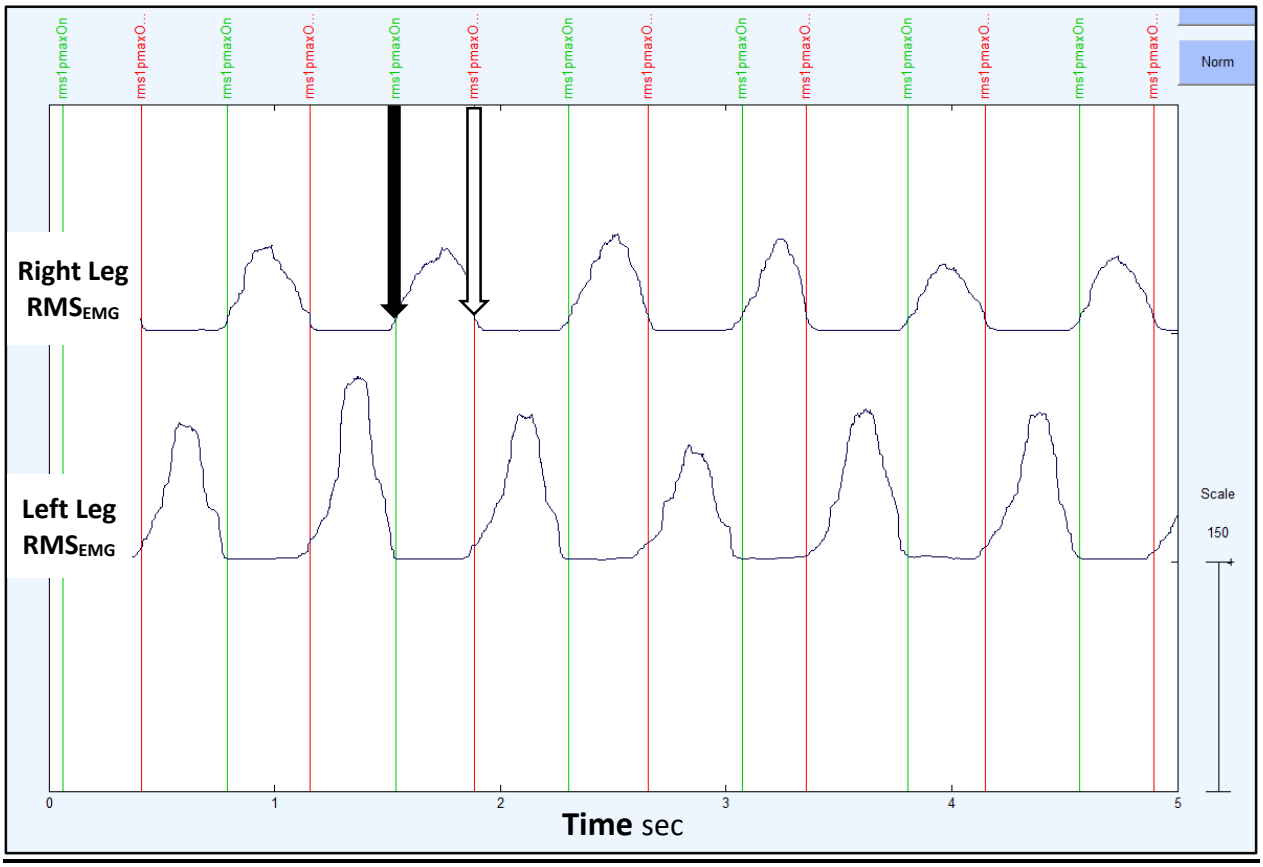

Figure 4. Example of a trace showing the RMS_{EMG} for the right (top) and left (bottom) legs over 5 seconds. EMG burst onset (black arrow) and offset (white arrow) markers are indicated, as an example, for the right leg only.

EMG threshold determination

A composite plot combining the RMS_{EMG} of each burst from both legs was constructed for each participant. This plot consisted of the RMS_{EMG} (in μV, with a single data point corresponding to each burst) plotted against the test duration (in seconds). A trimmed mean was then applied to this plot, whereby a moving average with a 30-point window and 10-point overlap was used, where the lowest 10 and highest 10 values in each window were discarded (trimmed) and an average of the median 10 points used

(Figure 5). The EMG_{Th} was then determined by a computer algorithm as the point of least residual sum of squares for any two linear regression-line divisions of the data, similar to the approach of Hug et al. (2006b)

Since the algorithm would always mathematically determine a point of least residual sum of squares, even if no true or discernible threshold exists, an additional criterion was used to qualify a threshold. Since EMG_{Th} was not expected to occur earlier than 70% of peak exercise intensity, a linear trend line was determined for the initial 70% of the test duration, with confidence intervals of 3 standard deviations above and below the trend line, extending to the end of the trace (Figure 5). To be regarded as an EMG_{Th}, the plot must have surpassed and stayed above the upper confidence limit (*e.g.*, Figure 5), without descending back to within the confidence interval until the end of the test (*e.g.*, Figure 6). In cases where a drop in the EMG signal was observed at the end of the test in conjunction with a fall in cadence below 80 rpm, the plot was truncated at the point where cadence began to fall. For those participants showing an EMG_{Th}, the identified point as a time (seconds) was converted to a power output (W) using a simple linear formula including the starting power output and ramp protocol. This EMG_{Th} power output was expressed as a percentage of the peak power output reached during the EMG_{Th} exercise test ($\%P_{max}$). The power output at the EMG_{Th} was then expressed as percentage of VO_2pk ($\%VO_2pk$).

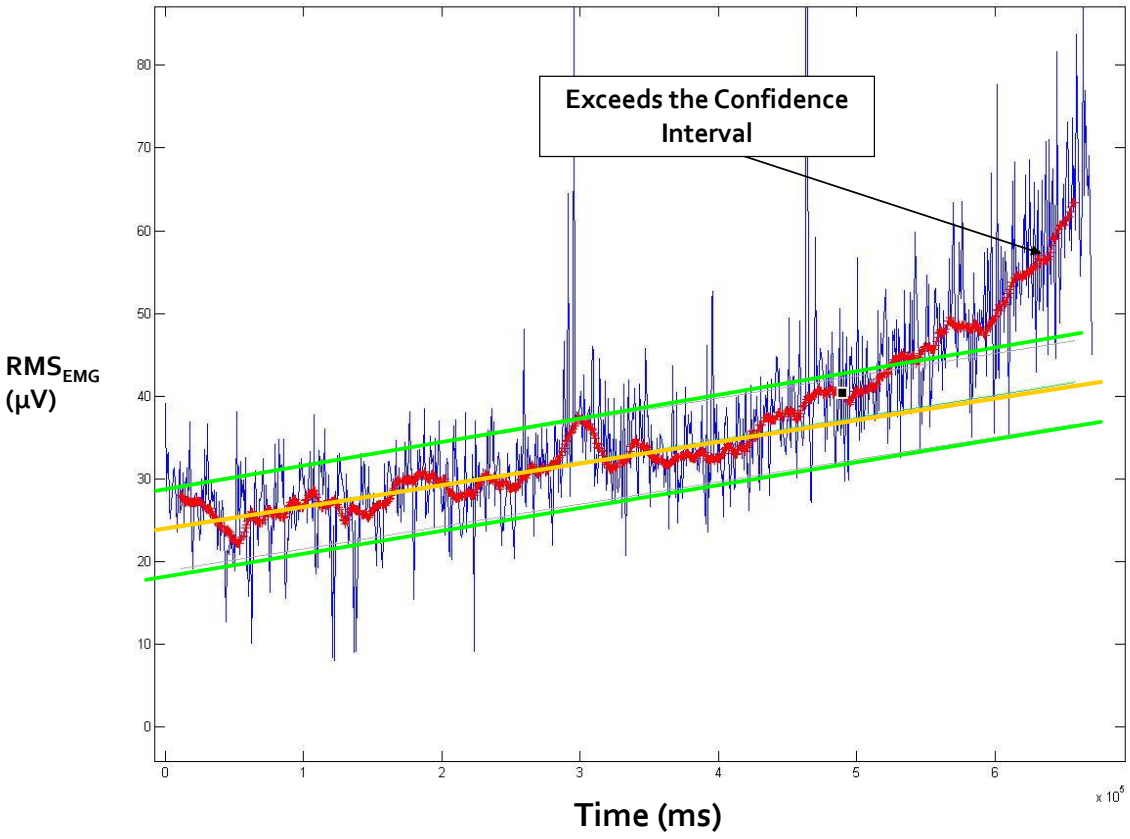

Figure 5. Sample EMG_{Th} trace demonstrating the *positive* criterion for EMG_{Th} identification. Black square (■) = mathematically-determined EMG_{Th} point, ▬ = raw data points, ▬ = trimmed mean, ▬ = trend line for 70% of the data trace, ▬ = confidence interval ±3 SDs above/below the trend line, based on the first 70% of the trace.

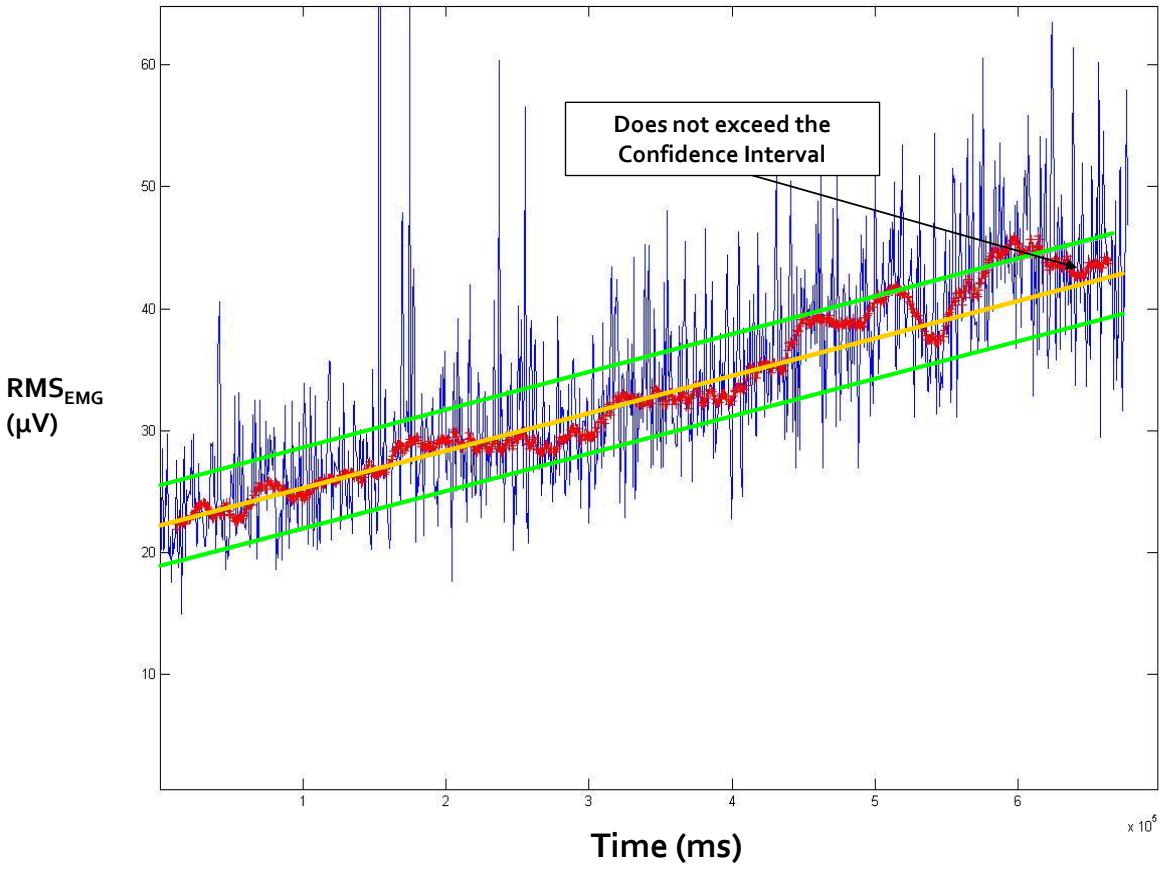

Figure 6. Sample trace demonstrating the *negative* criterion for EMG_{Th} identification. No EMG_{Th} is identifiable in this trace. ▬ = raw data points, ▬ = trimmed mean, ▬ = trend line for 70% of the data trace, ▬ = confidence interval of ±3 SDs above/below the trend line, based on the first 70% of the trace.

Statistical Analysis

All statistical analysis was performed using SPSS v.20 (SPSS Inc., Chicago, IL). The data for all groups are presented as means (M) ± 1 standard deviation (SD). All data were normally distributed. Group differences in the $\%P_{max}$ and $\%VO_{2max}$ at which the EMG_{Th} occurred were assessed using a two-tailed, homoscedastic Student's t-test. Pearson's Correlations were computed between the EMG_{Th} (both as $\%VO_2pk$ and $\%P_{max}$)

and VO_2pk/kgBW, PO_2pk/kgBW, P_{max}/kgBW, leisure time physical activity, and training hours per week, respectively. The acceptable level of significance for all tests was set at $p<.05$.

Results

Table 1 lists some of the relevant participant characteristics. Boys were significantly smaller than men but no differences were observed in body composition or maximal aerobic capacity. Sexual maturity level in the boys ranged from stages 1 to 4 on the Tanner scale. Of the twenty-three boys tested, eight were self-assessed as Tanner 1, eight as Tanner 2, five as Tanner 3, two as Tanner 4, and one participant was excluded due to concerns over misreport. Years to age of Peak Height Velocity (PHV) for the boys averaged 2.30±0.63 years. VO_2 data was unavailable for one participant from the boys' group.

Table 1. Participants' physical characteristics and peak oxygen consumption

	Men	**Boys**
n	21	23
Age (yrs)	23.4 (4.1)	11.1 (1.1)*****
Mass (kg)	75.4 (10.4)	37.1 (7.5)*****
Height (cm)	181.5 (6.3)	145.7 (8.6)*****
% Body Fat	14.0 (3.6)	14.2 (3.2)
G-S Activity Score	79.1 (50.9)	81.6 (25.3)
Sport training (hours/week)	5.6 (4.8)	5.4 (2.6)
VO_2pk (ml/kg/min)	49.7 (5.5)	50.1 (7.4)
RER at VO_2pk	1.15 (0.06)	1.05 (0.08)*****
HR at VO_2pk (bpm)	194 (7)	198 (11)

Values are Means (±1SD). ***** indicates significant difference between groups, $p<.05$

An EMG_{Th} was identified in 20 out of the 21 men (95.2%), whereas 18 of the 23 boys (78.3%) showed an EMG_{Th} ($\chi^2_{(1, n=44)} = 2.69$, $p = .10$). Group differences between the boys and men who *did* demonstrate an EMG_{Th} showed a later (higher) threshold in the boys compared with the men in terms of $\%P_{max}$ (86.3±9.6% *vs.* 79.7±10.0%, respectively; t (36) =-2.08, $p < .05$) and a trend for a higher threshold in the boys *vs.* men in terms of $\%VO_2pk$ (101.2±11.4 *vs.* 94.8±12.2%, respectively; t (35) = -1.62, $p = .12$) (Figure 7).

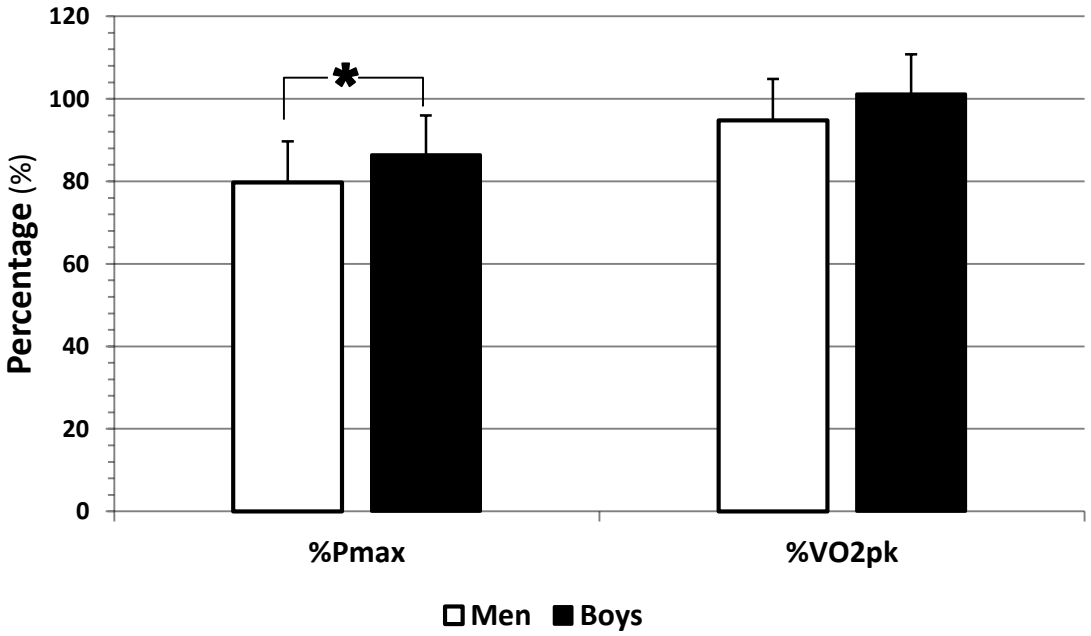

Figure 7. Group differences in the relative exercise intensity at EMG_{Th} between boys and men who *did* demonstrate an EMG_{Th}. Values are Means ±1SD. * = boys significantly higher than men, $p<.05$.

Correlational analyses (Table 2) revealed that for all participants, EMG_{Th} as $\%VO_2pk$ was significantly negatively correlated with PO_2pk/kgBW ($r = -.39$, $p < .05$). For the men's group, no significant correlations were found between EMG_{Th} (both as $\%P_{max}$ and $\%VO_2pk$) and VO_2pk, specific P_{max} (W/kgBW), or specific PO_2pk (W/kgBW), leisure

time physical activity, or training hours/week. For the boys' group, there was a significant moderately-negative correlation between EMG_{Th} as $\%VO_2pk$ and $VO_2pk/kgBW$, (r = -.50, p <.05), as well as $PO_2pk/kgBW$ (r= -.52, p<.05).

Table 2. Correlations between the EMG_{Th}, normalized PO_2pk and VO_2pk, leisure time physical activity, and sport training in boys and men who *did* show an EMG_{Th}

	Boys		**Men**		**All**	
	EMG_{Th} $\%P_{max}$	**EMG_{Th} $\%VO_2pk$**	**EMG_{Th} $\%P_{max}$**	**EMG_{Th} $\%VO_2pk$**	**EMG_{Th} $\%P_{max}$**	**EMG_{Th} $\%VO_2pk$**
Specific PO_2pk (W/kg)	-.16	**-.52 ***	-.11	-.23	-.17	**-.39 ***
Specific P_{max} (W/kg)	-.10	-.38	.01	.16	-.10	-.13
VO_2pk (ml/kg/min)	-.22	**-.50***	.03	.01	-.06	-.22
G-S Activity Score	-.03	-.17	-.05	-.07	-.02	-.07
Sport Training (hours/week)	-.07	.06	-.07	-.11	-.07	-.07
Values are Pearson's Correlations, *r*. ***** = significant correlation, p <.05.						

The boys' group was subdivided into those who *did* demonstrate an EMG_{Th} (positive) and those who *did not* (negative) for further analysis.

In the boys group, the 18 boys who *did* show an EMG_{Th} had significantly longer test duration than the 5 boys who *did not* show an EMG_{Th} (Table 3). There were no significant differences between the boys' groups in terms of age, maturity, anthropometric measures, VO_2pk, training hours/week, or leisure time physical activity.

Table 3. Comparison between boys who did and did not show an EMG_{Th}

	Positive (showed EMG_{Th})	**Negative** (no EMG_{Th})
n	18/23 (78.3%)	5/23 (21.7%)
Age (years)	11.1 (1.2)	11.1 (0.8)
Years to Age of PHV	2.26 (0.68)	2.52 (0.32)
Mass (kg)	38.0 (7.9)	34.0 (5.0)
Height (cm)	146.8 (9.0)	141.7 (6.4)
% Body Fat	14.3 (3.3)	13.8 (3.3)
Lean Body Mass (kg)	32.4 (6.2)	29.2 (3.8)
Vastus lateralis Muscle Volume (cm^3)	30.9 (7.7)	27.4 (4.6)
G-S Activity Score	83.5 (27.3)	74.9 (16.9)
Sport training (hours/week)	5.3 (2.5)	5.9 (3.5)
VO_2pk (ml/kg/min)	50.5 (7.8)	48.9 (6.1)
EMG_{Th} Test Duration (min)	10.0 (1.1)	8.4 (1.2)*****

Values are Means (±1SD). ***** = $p < .05$, significant difference between group.

In the men's group, the one man who did not show an EMG_{Th} had very high VO_2pk/kgBW and specific PO_2pk/kgBW, showing values roughly 2.5 SDs above the mean.

Discussion

One of the main findings of this study was that 78.3% of the boys demonstrated an EMG_{Th} compared with 95.2% of the men, though this difference was not statistically significant ($\chi^2_{(1, n=44)} = 2.69$, $p = .10$). Under the differential motor unit activation hypothesis, less boys than men might be expected to demonstrate an EMG_{Th} assuming boys lack the large, synchronous recruitment of type-II MUs attributed to the EMG_{Th}. However, it could be that the boys who *did not* show EMG_{Th} utilized other muscles to a greater extent to generate the power output, such that a large increase in type-II MU recruitment from *vastus lateralis* did not occur. Even so, among the boys and men who *did* present an EMG_{Th}, the boys' EMG_{Th} occurred at a significantly higher $\%P_{max}$ and tended to be higher as $\%VO_2pk$ than the men's.

Further analysis was performed with the assumption that in participants who *did not* demonstrate an $EMG_{Th,}$ the EMG_{Th} would potentially occur at or beyond the end of their respective tests (*i.e.,* at 100% P_{max}). Thus, for these participants, the EMG_{Th} was defined as occurring at 100% P_{max}. Under this definition and expressed as $\%P_{max}$, the boys' EMG_{Th} was further apart from that of the men, (89.1±10.5 *vs.* 80.7±10.7% $P_{max,}$ respectively; $t(42) = -2.73$, $p<.05$), while the trend for a higher $EMG_{Th,}$ as $\%VO_2pk$ was stronger (102.1±10.8 *vs.* 95.7±12.5% VO_{2pk}, respectively; $t(41) = -1.81$, $p = .08$).

There is reason to assume that participants who *did not* demonstrate an EMG_{Th} were not able to attain sufficiently high force and power output under the employed exercise protocol to elicit the threshold. During a progressive cycling test, the force produced by the muscles to push the pedals increases with the progressively increasing power output. However, Sargeant et al. (1985) demonstrated that even at the end of a

progressive cycling test to exhaustion, corresponding to an exercise intensity of 100% VO_2pk, at a fixed cadence of 70rpm, the muscles produced only 51±9% of the maximum force generated during a 20-second peak cycling power test at the same pedalling cadence. Activation of type-IIAB (IIA-IIX) and IIB (IIX) motor-units during progressive cycling exercise has been demonstrated in adults, at an exercise intensity as high as 91% VO_2pk (Vollestad & Blom 1985), an exercise intensity that would only require ~50% of the potential maximal force exerted on the pedals, as demonstrated by Sargeant et al. (1985). Therefore, assuming that EMG_{Th} can occur at or above 50% of the maximum force, the EMG_{Th} would only be detected in those participants who were able to exceed this force during the progressive exercise protocol. The boys who *did not* show an EMG_{Th} likely ended their cycling test before achieving a high enough power output to elicit the muscle to produce a high enough force needed to observe the EMG_{Th}. Thus, defining the EMG_{Th} as 100% P_{max} for those participants may be considered an underestimate of what their respective EMG_{Th} might actually be, had they been able to reach higher finishing power outputs. In spite of this underestimate, the boys' mean EMG_{Th} as $\%P_{max}$ was still significantly higher than the men's. Furthermore, the differences reported between the boys and men who *did* show EMG_{Th} ('positive') might also justifiably be considered underestimates since those participants who did *not* show ('negative') were excluded. Taken together, these findings suggest that boys activate their type-II motor-units to a lesser extent than men. Along with the well-established child–adult differences in muscular performance during exercise, metabolic responses to exercise, and neuromuscular function discussed earlier, the results provide further support for the hypothesis of child–adult differential motor-unit activation.

To our knowledge, this is the first study to investigate the EMG_{Th} in children. The results for our men's group are in close agreement with previous findings. Hug et al. (2006a), using a similar cycling protocol, found the EMG_{Th} to occur at 78±11% P_{max} in the *vastus lateralis* of sedentary men, similar to our value of 79.7±10.0% P_{max} for the men's group. Likewise, using a similar exercise protocol to our own, Takaishi et al. (1992) found men's EMG_{Th} to occur at ~90% VO_2pk, in close agreement with our finding of 94.8±12.2% VO_{2pk} for the men's group, while Lucia et al. (1999) found an EMG_{Th} at 90.2±6.9% VO_{2max} (VO_2pk), albeit in elite cyclists.

As could have been expected from other forms of threshold, the boys showed a significantly higher relative EMG_{Th} than the men, expressed as $\%P_{max}$, and a trend for a higher EMG_{Th} expressed as $\%VO_2$pk. Given the association between the EMG_{Th} and both the lactate and ventilatory thresholds observed in men, it could be expected that the EMG_{Th} in boys would occur at similar relative exercise intensities as the lactate and ventilatory thresholds. However, the EMG_{Th} occurred at 101.2±11.4% VO_2pk in our boys' group. This is a much higher relative exercise intensity than typical for the lactate and ventilatory thresholds (*cf.* Pfitzinger & Freedson 1997). For example, Klentrou et al. (2006) found boys' ventilatory threshold to occur at 64.9±7.1% VO_2pk, while Anderson & Mahon (2007) observed the ventilatory and lactate thresholds at 67.9±2.6% and 63.9±5.0%VO_2pk, respectively. This apparent discrepancy is likely due to the fact the metabolic acidosis and increased lactate production can be brought about by the increasing exercise intensity of type-I motor-units with or without type-II motor-unit involvement, while the EMG_{Th} is entirely dependent on the latter. Furthermore, while the

lactate and ventilatory thresholds are detected systemically, EMG_{Th} is detected within an individual muscle.

There is an apparent discrepancy between the intensity at which our boys' EMG_{Th} occurred and those previously reported for boys at other forms of threshold. However it is interesting to note that the differences in the EMG_{Th} (as $\%VO_2pk$) between the boys and men was 6.4% for those participants who *did* show an EMG_{Th}. This difference is comparable to previously reported differences between boys and men in the ventilatory threshold (7.2%, Klentrou et al. 2006; 8.9%, Anderson & Mahon 2007) and the lactate threshold (9.1%, Anderson & Mahon 2007) during cycling exercise. Results from studies comparing the EMG_{Th} intensity between different groups of men are slightly at variance with the results of this study. Tikkanen et al. (2012) found EMG_{Th} (as $\%VO_2pk$) to occur 15.2% higher in endurance-trained compared with recreationally-active participants during treadmill running (96.4 *vs.* 81.2%, respectively), while Chwalbinska-Moneta et al. (1994) found EMG_{Th} in their endurance-trained participants to be 4.4% higher ($\%VO_2pk$) and 12.4% higher ($\%P_{max}$), than in sprint-trained athletes. Thus, it appears that in men, endurance training can produce larger differences in the relative EMG_{Th} intensity than those shown between the boys and men in the present study. It is important to note, however, that the boys–men differences in the present study are likely an underestimate because the 5 boys who did *not* show EMG_{Th} were excluded. Assuming that EMG_{Th} would occur at 100% P_{max} or higher in the boys, the boys–men difference in the present study would be expected to be larger and more similar to the between-group differences discussed above.

Recognizing the possible confounding effects of endurance training, it was initially intended to exclude, or separately consider endurance-trained individuals. However, no correlation could be found between EMG_{Th} intensity and any training indices in either the boys' or the men's group. Hence, it was decided, to pool trained and untrained participants in their respective boys or men groups.

By virtue of its characteristically low-intensity, endurance-training primarily or exclusively engages and trains type-I MUs, making endurance-trained individuals functionally more reliant on that MU type. This is akin to having a greater type-I MU composition, or as is allegedly the case with children, having a more limited use of type-II MUs. Thus, in terms of MU-type use, children mimic the endurance athlete profile and could thus be expected to possess superior muscle endurance capacity compared with untrained adults, as has already been previously shown (Armatas et al. 2010; Kotzamanidou et al. 2005; Paraschos et al. 2007; Zafeiridis et al. 2005; Halin et al. 2003).

To illuminate factors which could possibly determine whether EMG_{Th} would occur in some participants, but not others, the boys' group was subdivided along this dichotomy (Table3). Boys who demonstrated an EMG_{Th} ("positive") had longer mean test durations than those who did not ("negative"). One explanation for this finding is that boys who could last longer in their respective tests were able to reach somewhat higher power outputs, high enough to detect their respective EMG_{Th}. At the end of a ~10-min. test to exhaustion, muscle force applied to the pedals would be ~50% of the maximum available force at the same pedalling cadence (Sargeant et al. 1985; Sargeant & Jones 1995) which would suggest that additional muscle capacity and potential MU recruitment has not been accessed. The boys who *did not* demonstrate an EMG_{Th} were thus likely

unable to attain sufficiently high power outputs for their threshold to manifest itself. Indeed, the boys who *did not* show an EMG_{Th} tended to have a lower P_{max} than the boys who showed EMG_{Th} (132 *vs.* 152 W, respectively), although this difference was not significant due to the small size of the "negative" group (n = 5; p =.27). It therefore appears that the ability to attain higher power outputs increased the likelihood of exhibiting an EMG_{Th}. The negative correlations between the boys' $EMG_{Th,}$ PO_2pk/kgBW, and VO_2pk/kgBW support this explanation (Figures 8 & 9). That is, boys with higher aerobic capacity were able to reach higher maximal power outputs, high enough to elicit EMG_{Th} in the first place and to make it appear at lower relative intensities if they had reached it earlier. Thus, higher aerobic fitness seems to paradoxically be associated with a lower (earlier) EMG_{Th}, making those boys with higher aerobic fitness more similar to men. Additionally, the boys who *did not* show an EMG_{Th} tended to have lighter body weight, lower pubertal (Tanner) maturation rating, and more years to the age of PHV. It could thus be suggested that the EMG_{Th} "negative" participants were slightly less mature than the EMG_{Th} "positive" participants, however this suggestion needs to be examined in a larger sample.

This inverse VO_2pk-EMG_{Th} relationship was not evident in the men. The reason for this likely lies in the fact that men have higher glycolytic capacity (Berg et al. 1986; Eriksson et al. 1973; Kaczor et al. 2005), presumably related to their greater capacity for

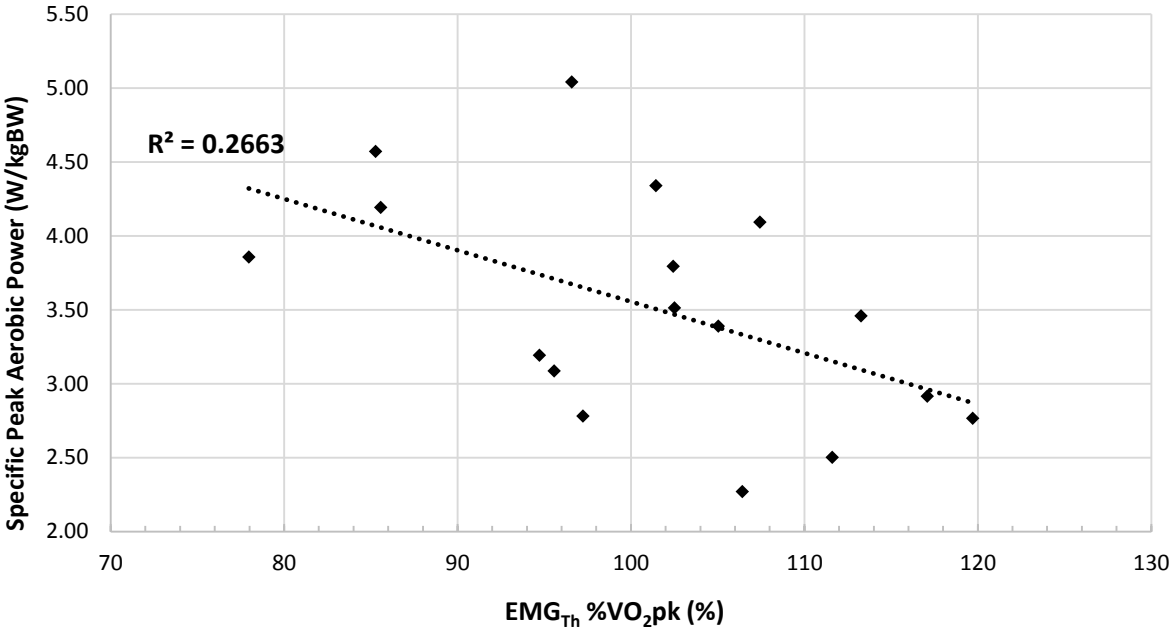

Figure 8. Correlation between specific PO_2pk and EMG_{Th} as %VO_2pk for the boys who *showed* EMG_{Th}.

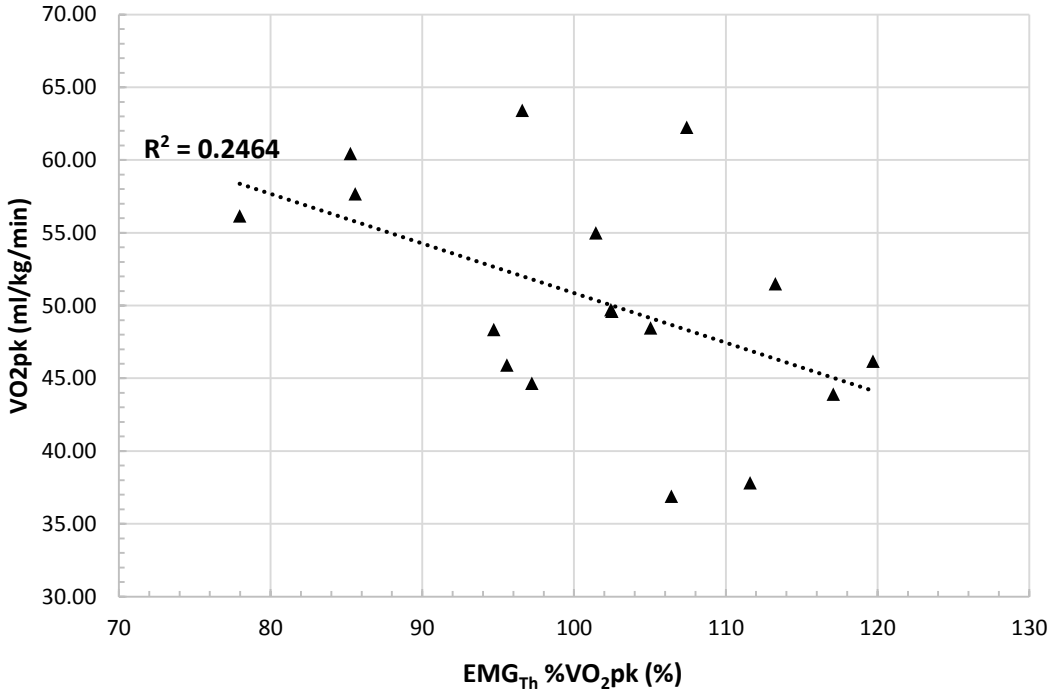

Figure 9. Correlation between VO_2pk and EMG_{Th} as %VO_2pk for the boys who *showed* EMG_{Th}.

type-II MU recruitment. This enables them to attain higher relative muscle forces in the EMG_{Th} test, thus substantially increasing their likelihood of reaching the intensity and type-II MU recruitment necessary for manifesting an EMG_{Th}. In other words, the men's maximal power output is not as dependent on aerobic capacity as it is in the boys' case. Thus, this observation lends additional support to the differential MU activation hypothesis.

In the men's group, only one participant did *not* show an EMG_{Th}. This participant showed disproportionately high relative (per kgBW) VO_2pk (63.6 ml/kg/min) and PO_2pk (5.14 W/kg). Barring possible EMG_{Th} misidentification, this participant's superior aerobic capacity, possibly the result of exceptionally high type-I fibre composition and correspondingly low glycolytic capacity, enabled him to rely on type I MUs to the within the test's attained intensity range. Thus, highly endurance-trained athletes and/or individuals with exceptionally-high type-I fibre composition, may be mimicking children who allegedly are functionally limited in recruiting the type-II MUs.

In summary, the findings of the study are particularly relevant to the differential motor-unit activation hypothesis. The EMG_{Th} is believed to reflect a large and synchronous increase in type-II motor-unit recruitment. Thus, the absence of an EMG_{Th} in some boys would be expected with a lesser activation of type-II motor-units. The finding that the majority of both boys and men demonstrated an EMG_{Th}, along with the qualitative similarity in the EMG_{Th} response to increasing exercise intensity in both groups, could suggest that the pattern of motor-unit recruitment is similar between boys and men during progressive cycling exercise. Quantitatively, however, the boys' EMG_{Th} occurred at a higher $\%W_{max}$ and demonstrated a trend to occur at a higher $\%VO_2pk$ than

the men, suggesting a later recruitment of type-II motor-units in boys. This is in line with previous findings of higher relative lactate and ventilatory thresholds in boys compared with men and is to be expected given that boys' higher endurance type-I motor-units would be utilized relatively longer, before their type-II MUs are recruited.

Limitations

A possible limitation of the present study was the age of the boys' group. With a mean age of 11.1±1.1 years, it is possible that many of the boys may have already reached significant levels of neuro-motor maturity. Grosset et al. (2008) showed that the differences in *triceps surae* neuromuscular efficiency, level of activation following supramaximal electrical stimulation, and the activation deficit between children and adults diminishes with age, with 11-year-old children's values already approaching those typical of adults. Similarly, some evidence suggests that a type-I to type-II shift in muscle fibre-type composition will have, by age 11, already attained muscle-fibre composition similar to 15–19-year-old adolescents (Jansson 1996). Thus, a younger cohort might have shown still greater differences from men.

Another limitation is that the present study measured neither MVC, nor the force exerted per pedal stroke throughout the EMG_{Th} exercise test. Such measurements would be beneficial in determining the percentage of MVC force being produced at the EMG_{Th} workload, as well as at the highest workload achieved, in order to better assess whether differences in potential muscle capacity exist between boys and men.

Although the EMG_{Th} is widely regarded as reflecting the onset of increased type-II motor-unit recruitment, that concept is still unproven. Thus, the main limitation of the

present study is that its conclusions, regarding child–adult differential MU activation, are conditional on the veracity of the EMG_{Th} concept.

To our knowledge, there are currently no means of direct identification of individual motor-units in a manner that can enable reliable conclusions about the entire MU pool. Moreover, any invasive procedure that could possibly allow that will likely be off limits to children due to ethical considerations.

Future directions

A parallel, non-invasive approach could be EMG frequency analysis, such as the mean power frequency and recent, more sophisticated, frequency analysis techniques (von Tscharner 2000; Wakeling et al. 2002). A related approach might be based on the idea that motor units are roughly type-compartmentalized in specific fixed locations along a given muscle (Wakeling 2009). This could potentially allow for site-specific EMG monitoring and comparisons under increasing exercise intensities.

Follow-up studies examining EMG_{Th} occurrence in multiple leg muscles simultaneously (*i.e.*, all the *quadriceps* and hamstring muscles, as well as the ankle flexors and extensors) would be beneficial in determining whether the observed child–adult difference in EMG_{Th} could be attributed to child–adult differences in whole muscle recruitment strategies during progressive exercise. Future directions also include investigating the EMG_{Th} in girls compared with women since female child–adult exercise-related differences are typically less extreme than those seen in males (*e.g.*, Falk et al. 2009a). Examining EMG_{Th} occurrence across different maturity groups would also help to expand the results of the current study and further our understanding of how both sex and maturity may influence how neuromuscular activation changes with growth and

development. Perhaps, with the use of more advanced technology (*e.g.*, MRI) or higher resolution EMG using electrode array techniques, a more direct examination of specific motor-unit activation pattern could be carried out in future studies.

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Appendix A: Tables

Table 4. Child–adult differences in neuromuscular function, muscular performance, and metabolic responses during exercise. (Dotan et al. 2012)

Muscular Performance Characteristics	**In Children (*vs.* Adults)**
Maximal Isometric Strength	Lower
Short-term Power	Lower
Recovery of Power	Faster
Muscle Endurance	Higher
Force Kinetics	Slower
Metabolic Responses to Exercise	
Peak [La]	Lower
PCr Recovery	Faster
Pi/PCr Threshold	Higher
Fat Oxidation	Higher
VO_2 kinetics	Faster
Threshold Responses to Exercise	
La & Ventilatory Thresholds	Higher (as $\%VO_{2max}$)
Neuromuscular Function	
Rate of EMG rise in first 30ms following neural stimulation (Q_{30})	Lower
Electromechanical Delay	Longer
Mean Power Frequency (MPF)	Lower
Motor-unit Activation (Interpolated-twitch)	Lower

Table 5. **Relationship between EMG and metabolism-related thresholds**

Reference	Participants	Exercise Mode	Metabolic Indices	Main Finding
Chwalbinska-Moneta et al. Clin J Sports Med. 4: 31-38, 1994.	7 endurance-trained (19.7±1.1 yrs), 7 speed-trained male athletes (19.6±1.0 yrs)	Cycling	Blood La threshold, anaerobic threshold	EMG threshold for m. soleus occurred at the same exercise intensity as the individual anaerobic threshold in both endurance-trained and speed trained athletes; EMG threshold for m. rectus femoris occurred at the same intensity as the blood La threshold (>4 mM).
Chwalbinska-Moneta et al. J Physiol Pharmacol. 49 (3): 433-441, 1998	28 male soccer players (20.6±0.8 yrs, mean VO_{2max}: 4.04±0.12 L/min.)	Cycling	Blood La, plasma noradrenaline and adrenaline thresholds	EMG derived from the soleus and rectus femoris muscles was closely correlated with blood [La]; EMG threshold exercise intensities did not differ significantly from the blood lactate, plasma noradrenaline, and adrenaline thresholds.
Hug et al. Eur J Appl Physiol. 90: 643–646, 2003.	8 professional road cyclists (24 yrs)	Cycling	Ventilatory thresholds	The first EMG threshold occurred significantly before the first ventilatory threshold (52% and 62% of peak power output, respectively). No significant difference between the second EMG threshold and the second ventilatory threshold (86% and 89% of peak power output, respectively).

Lucia et al. Br J Sports Med. 33:178–185, 1999.	28 elite male road cyclists (24±4yrs)	Cycling	La threshold, OBLA, ventilatory thresholds	No significant differences between the first EMG thresholds in m. vastus lateralis, m. rectus femoris, the first ventilatory threshold, and the La threshold (62.8%, 69%, 64.6%, 68.7% of VO_{2max}, respectively). No significant differences between the second EMG thresholds in m. vastus lateralis, m. rectus femoris, the second ventilatory threshold, and OBLA (86.9%, 88%, 84.6%, 87.7% of VO_{2max}, respectively).
Maestu et al. JSCR. 20(4): 824–828, 2006.	9 national-level male rowers (21.8±4.4 yrs)	Rowing	Ventilatory thresholds	No significant difference between the EMG threshold and the second ventilatory threshold (occurred at 258.89±27.13W and 248.9 ±26.67W power outputs, respectively).
Moritani et al. Am. J. Phys. Med. 63 (3), 122-13, 1984	5 male subjects (24.8±5.4 yrs)	Forearm contractions	Venous blood La, venous La threshold	High correlation between venous blood [La] and iEMG; abrupt increase in iEMG and decrease in MPF at the onset of the venous lactate threshold.

Moritani et al. J. Appl. Physiol. 74(4): 1729-1734, 1993	8 male and 12 female subjects (21±2.3 yrs); fitness ranged from trained track & field athletes (4 male, 6 female) to untrained sedentary individuals.	Cycling	Anaerobic threshold	The neuromuscular fatigue threshold was highly correlated with the anaerobic threshold, but occurred at a significantly greater exercise intensity (1.84±.55 L/min *vs.* 1.72±.54 L/min VO_2, respectively).
Nagata et al. JJP. 31: 585-597, 1981.	10 healthy male college students (21±1.6 yrs)	Cycling	Blood anaerobic threshold (arterial blood lactate, P_{O2}, P_{CO2}, HCO_3^-, pH), Ventilatory threshold	VO_2 at which the iEMG threshold occurred (1.91 L/min) was significantly correlated with both the blood anaerobic threshold (1.71 L/min) and the ventilatory threshold (1.87 L/min).
Taylor & Bronks. Eur J Appl Physiol. 69: 508-515, 1994.	10 trained male subjects (20.18±1.03 yrs; mean VO_2max: 58.51±2.22 $ml.kg^{-1}. min^{-1}$1)	Treadmill Running	Blood La threshold, Ventilatory threshold	No significant difference between iEMG threshold and the ventilatory threshold (both occurred at ~79% of VO_2pk); iEMG threshold was significantly greater than the La threshold (79.76% *vs.* 72.17% VO_{2peak}).

Tikkanen et al. Physiol Meas. 33 (4): 603-14, 2012.	12 endurance runners, 15 recreationally active subjects (males, 25.1 ± 2.7 yrs)	Treadmill running	OBLA, Ventilatory thresholds	Significant correlation between EMG threshold and OBLA, second ventilatory threshold. No significant difference in the VO_2 at which OBLA, the second ventilatory threshold, and the EMG threshold occurred.

Appendix B: Anthropometric Measurements Data Collection Sheet

NAME: _____ TEST DATE (MM/DD/YYYY): _____

ID NUMBER: _____ GENDER: M / F AGE: _____

DATE OF BIRTH (MM/DD/YYYY): _____ DOMINANT ARM: R / L

SUBJECT HEIGHT (cm): _____ SEATED HEIGHT (cm): ___ (Table = 75.5 cm) _____

SUBJECT WEIGHT (kg): _____

BIA - BMI: _____ BIA – % BODY FAT: _____

THIGH LENGTH (cm): _____

TRIAL 1	TRIAL 2	TRIAL 3	MEDIAN

THIGH CIRCUMFERENCE (cm): _____

TRIAL 1	TRIAL 2	TRIAL 3	MEDIAN

SKINFOLD MEASUREMENT (mm):

SITE	TRIAL 1	TRIAL 2	TRIAL 3	TRIAL 4 (>1 mm diff)	MEDIAN
TRICEP					
SUBSCAP.					
BICEPS					

SUM OF SKINFOLDS (mm): SUM @2 S.F _____

(2 Skinfold sites = Subscap+Tricep) % BODYFAT _____

SKINFOLD MEASUREMENT OF THE THIGH

SITE	TRIAL 1	TRIAL 2	TRIAL 3	TRIAL 4 (>1 mm diff)	MEDIAN
ANTERIOR					
POSTERIOR					
MEDIAL					
LATERAL					

SUM OF SKINFOLDS (mm):

SUM @4 S.F _____

MUSCLE DIAMETER (mm)

MUSCLE	TRIAL 1	TRIAL 2	TRIAL 3	TRAIL 4	MEDIAN
VASTUS LATERALIS					

Inseam - Crank Length Chart

(from Smith, D., Searle, B., and Thomas, S. *The Racing Bike Book*. Haynes Publishing, 1997)

Inseam (cm)	Crank Length (mm)
82-86	170
78-82	165
74-78	160
70-74	155
66-70	150
62-66	145
58-62	140
54-58	135
50-54	130

Appendix C: Borg Rating of Perceived Exertion (RPE) Scale

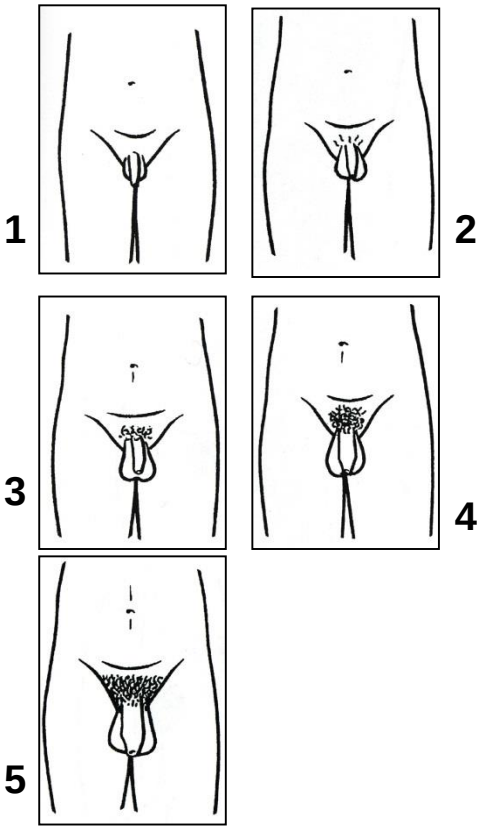
Appendix D: Pubertal Stage Questionnaire (Tanner, 1962)

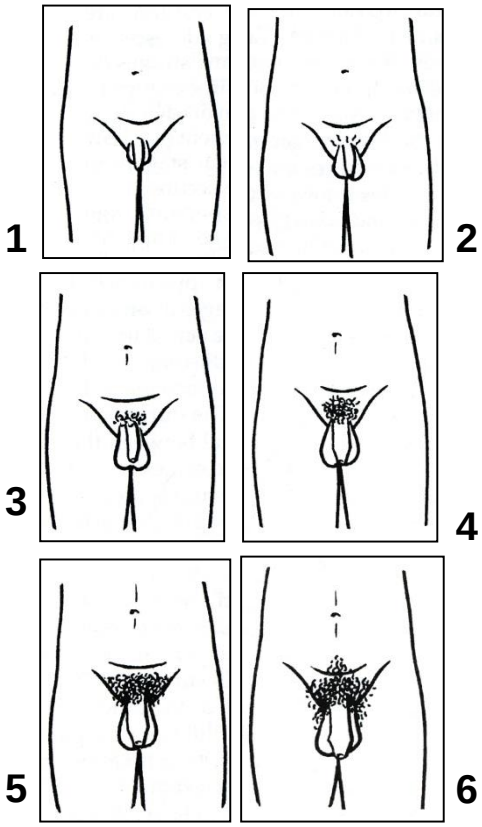
Male Pubertal Stage

This survey will be used to assess the maturational levels of the participant. For each photo choose the appropriate stage and place an X in the corresponding square.

ID: _____ Date: _____

• Please circle the box that looks most like you		• Please look at the pubic hair only • Please circle the box that looks most like you





Appendix E: Godin-Shepard Leisure Time Exercise Questionnaire

GODIN-SHEPHARD LEISURE-TIME EXERCISE QUESTIONNAIRE

1. Considering a **7-day period** (a week), how many times on the average do you do the following kinds of exercise for **more than 15 minutes** during your **free-time** (write on each line the appropriate number)?

(a) STRENUOUS EXERCISE (HEART BEATS RAPIDLY)
(*i.e.* running, jogging, hockey, football, soccer, squash, basketball,

cross country skiing, judo, roller skating, vigorous swimming,

vigorous long distance bicycling) **Times per week _____**

(b) MODERATE EXERCISE (NOT EXHAUSTING)
(*i.e.* fast walking, baseball, tennis, easy bicycling, volleyball,

badminton, easy swimming, alpine skiing, popular and folk dancing)

Times per week _____

(c) MILD EXERCISE (MINIMAL EFFORT)

(*i.e.* yoga, archery, fishing from river bank, bowling, horseshoes,

golf, snow-mobiling, easy walking)

Times per week _____

2. Considering a **7-day period** (a week), during your leisure-time, how often do you engage in any regular activity long enough to work up a sweat (heart beats rapidly)?

1. OFTEN	**2. SOMETIMES**	**3. NEVER/RARELY**
☐	☐	☐

Appendix F: Subject Screening and Medical History Questionnaire

APPLIED PHYSIOLOGY RESEARCH GROUP
DEPARTMENT OF KINESIOLOGY, BROCK UNIVERSITY

Your responses to this questionnaire are confidential and you are asked to complete it for your own health and safety. If you answer "YES" to any of the following questions, please give additional details in the space provided and discuss the matter with one of the investigators. You may refuse to answer any of the following questions.

Name: ——————————————— Date: ———————————————

1. Have you ever been told that you have a heart problem?

   YES NO

2. Have you ever been told that you have a breathing problem such as asthma?

   YES NO

3. Have you ever been told that you sometimes experience seizures?

   YES NO

4. Have you ever had any major joint instability or ongoing chronic pain such as in the knee, back or elbow?

   YES NO

5. Have you ever been told that you have kidney problems?

   YES NO

6. Have you had any allergies to medication?

   YES NO

7. Have you had any allergies to food or environmental factors?

   YES NO

8. Have you had any stomach problems such as ulcers?

YES NO

9. When you experience a cut do you take a long time to stop bleeding?

YES NO

10. When you receive a blow to a muscle do you develop bruises easily?

YES NO

11. Are you currently taking any medication (including aspirin) or have you taken any medication in the last two days?

YES NO

12. Is there any medical condition with which you have been diagnosed and are under the care of a physician (*e.g.*, diabetes, high blood pressure)?

YES NO

Appendix G: Training History Questionnaire

TRAINING HISTORY QUESTIONNAIRE FOR ATHELTES

Please fill in the table below to the best of your knowledge.

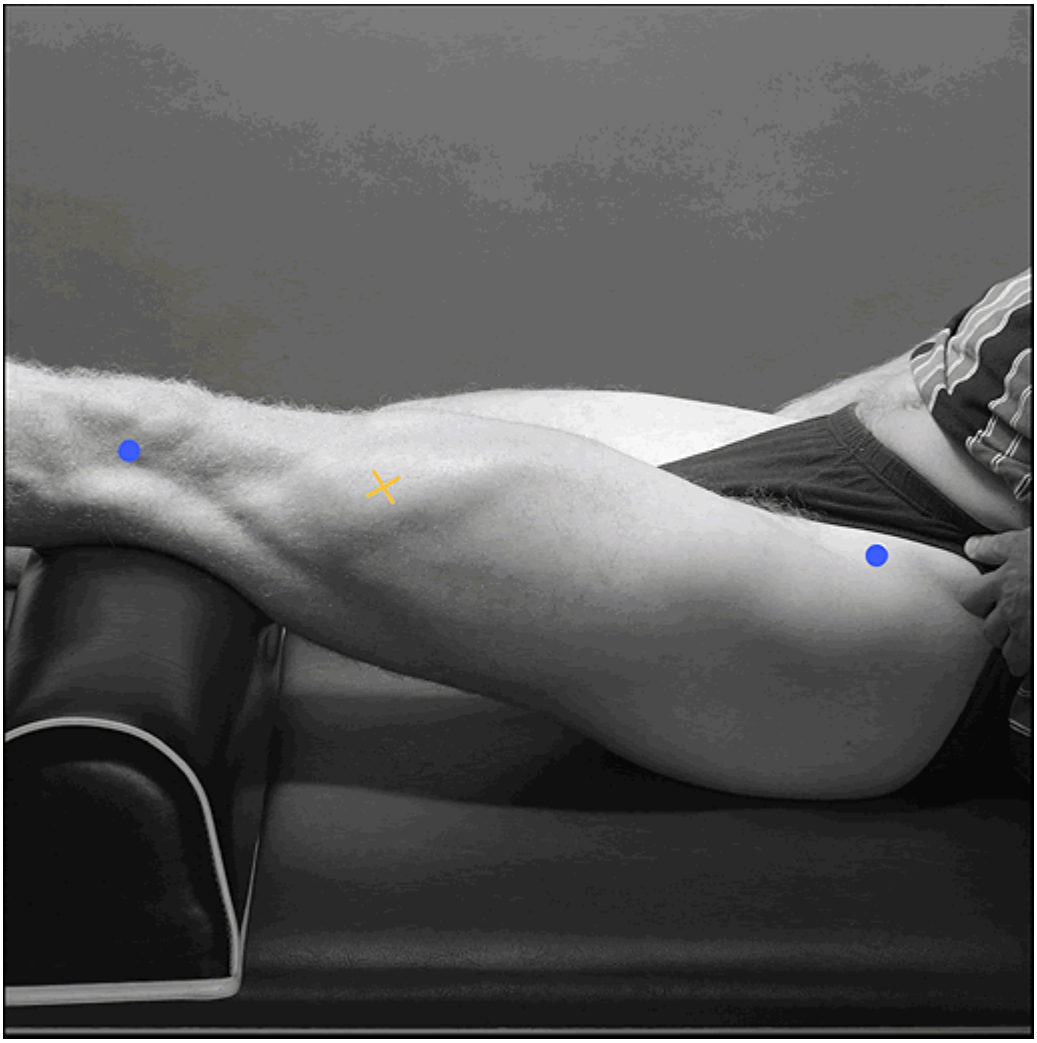
If you have any difficulties, discuss the matter with one of the investigators.

Activity/ Sport	**Level of Competition**	**# of years**	**Sessions /week**	**Min/ session**	**Intensity** (light, moderate, intense, very intense)	**Seasonal length**
Soccer						
Swimming						
Hockey						
Gymnastics						
Running						
Resistance						
Other						

Appendix H - Electrode Placement

From SENIAM "**Recommendations for sensor locations in hip or upper leg muscles**" (Available online from http://www.seniam.org/quadricepsfemorisvastuslateralis.html)

Muscle	
Name	Quadriceps Femoris
Subdivision	vastus lateralis
Muscle Anatomy	
Origin	Proximal parts of intertrochanteric line, anterior and inferior borders of greater trochanter, lateral lip of gluteal tuberosity, proximal half of lateral lip of linea aspera, and lateral intermuscular septum.
Insertion	Proximal border of the patella and through patellar ligament.
Function	Extension of the knee joint.
Recommended sensor placement procedure	
Starting posture	Sitting on a table with the knees in slight flexion and the upper body slightly bend backward.
Electrode size	Maximum size in the direction of the muscle fibres: 10 mm.
Electrode distance	20 mm.
Electrode placement	
- location	Electrodes need to be placed at 2/3 on the line from the anterior spina iliaca superior to the lateral side of the patella.
- orientation	In the direction of the muscle fibres
- fixation on the skin	(Double sided) tape / rings or elastic band.
- reference electrode	On / around the ankle or the proc. spin. of C7.
Clinical test	Extend the knee without rotating the thigh while applying pressure against the leg above the ankle in the direction of flexion.
Remarks	The SENIAM guidelines include also a separate sensor placement procedure for the vastus medialis and the rectus femoris muscle.



Appendix I – Physical characteristics, physical activity, VO2max and EMG-related results for men and boys (mean and SD)

	Men		Boys	
	Mean	**SD**	**Mean**	**SD**
Age_days	8559.048	1498.98831	4048.174	413.26622
Age_years	23.4334	4.10400632	11.0833	1.13146125
Weight_kg	75.42381	10.4314383	37.12609	7.46794467
Height_cm	181.481	6.25656607	145.6891	8.62013969
Seated_Height (cm)			151.9556	3.14684197
Sitting_Height (minus table)			76.45556	3.14684197
Leg_Length (cm)			69.89444	4.90215705
Years_PHV			-2.30303	0.63193295
Pubertal stage (pubic hair)	*n* each stage: Tanner 1 = 8 Tanner 2 = 8 Tanner 3 = 5 Tanner 4 = 2			
arm_Length (cm)	33.62857	1.67843464	26.9913	2.81649573
R_Thigh_Length (cm)	49.81429	2.53895367	39.38478	2.63763991
L_Thigh_Length (cm)	49.92143	2.60905018	39.55217	2.59051935
Mean_Thigh_Length (cm)	49.86786	2.44508618	39.46848	2.59278081
SF_Tricep (mm)	8.871429	2.34502513	9.847826	2.84491086
SF_Subscap (mm)	9.547619	1.87473173	5.904348	2.02450404
SF_Sum_2 Sites	18.41905	3.93568533	15.75217	3.52702161
for Slaughter Eqn	5.5	0	2.808696	0.82787408
SF_BF_2 (Slaughter, %)	13.95494	3.56026875	14.17119	3.23480319
SF_BF_2 (Slaughter, %)	13.95571	3.56212657	14.17174	3.23525136
BIA_PBF (%)	11.9381	3.6843556	11.57391	3.77349818

BMI (calculated)	22.86269	2.75046301	17.31667	1.77841384
LBM (calculated, %)	64.6414	7.45801568	31.739	5.82155353
R_SF_Ant_Thigh (mm)	9.280952	2.89579334	12.17826	4.40825223
R_SF_Post_Thigh (mm)	10.64412	4.37192329	14.10227	4.2731297
R_SF_Med_Thigh (mm)	10.85476	4.84321445	14.85	5.04950493
R_SF_Lat_Thigh (mm)	7.666667	2.69617012	11.31739	4.57718176
R_SF_Mean_Thigh (mm)	9.642063	3.22747641	13.21522	4.40884467
R_SF_Mean_Thigh (cm)	0.964206	0.32274764	1.321522	0.44088447
R_Thigh_Circumference (cm)	52.36667	4.06021346	39.35217	3.94563851
R_Thigh_Radius (cm)	8.334414	0.64620304	6.263093	0.62796787
R_Thigh_Lean_Radius (cm)	7.852311	0.58925926	5.602332	0.531685
R_Thigh_Lean_CSA (cm)	194.7457	28.3046344	99.4519	18.9816562
R_Muscle_Diameter (VL)	25.91393	4.43156283	19.56609	2.16901473
R_Muscle_CSA (VL)	5.421095	1.88454075	3.042097	0.65000801
R_Muscle_volume (VL)	64.52835	20.9647992	30.04729	7.4581764
L_SF_Ant_Thigh	9.738095	2.99624368	12.47391	4.59210958
L_SF_Post_Thigh	10.12222	5.02903985	12.59545	4.62927818
L_SF_Med_Thigh	11.9881	4.76014456	16.55652	5.81991691
L_SF_Lat_Thigh	7.65	3.06471858	12.01304	4.57207973
L_SF_Mean_Thigh (mm)	9.890873	3.43074149	13.54312	4.84414292
L_SF_Mean_Thigh (cm)	0.989087	0.34307415	1.354312	0.48441429
L_Thigh_Circumference	51.98571	4.14599635	39.11304	3.88134283
L_Thigh_Radius	8.273783	0.65985581	6.225034	0.6177349
L_Thigh_Lean_Radius (cm)	7.77924	0.59389242	5.547878	0.48587661
L_Thigh_Lean_CSA (cm)	191.1737	28.2701422	97.40435	17.17973
L_Muscle_Diameter (VL)	25.71467	5.14030926	19.45152	2.00751624
L_Muscle_CSA (VL)	5.391041	2.1186127	3.001922	0.60908753

L_Muscle_volume (VL)	65.37487	24.6690618	30.2765	8.05890053
Mean_SF_Ant_Thigh (mm)	9.509524	2.88338425	12.32609	4.45698812
Mean_SF_Post_Thigh (mm)	10.4625	4.58028263	13.34886	4.35335446
Mean_SF_Med_Thigh (mm)	11.42143	4.7427727	15.70326	5.33411674
Mean_SF_Lat_Thigh (mm)	7.658333	2.82536871	11.66522	4.47920536
Mean_SF_Mean_Thigh (mm)	9.754563	3.29066128	13.37917	4.59455251
Mean_SF_Mean_Thigh (cm)	0.975456	0.32906613	1.337917	0.45945525
Mean_Thigh_Circumference (cm)	52.17619	4.07285585	39.23261	3.88097366
Mean_Thigh_Radius (cm)	8.304099	0.64821514	6.244064	0.61767614
Mean_Thigh_Lean_Radius (cm)	7.81637	0.58554352	5.575105	0.50214201
Mean_Thigh_Lean_CSA (cm)	192.9635	27.9800984	98.40405	17.856382
Mean_Muscle_Diameter (VL) (mm)	25.8143	4.69709558	19.5088	1.94058623
Mean_Muscle_CSA (VL) (cm)	5.398749	1.96864159	3.017465	0.5808771
Mean_Muscle_volume (VL) (cm^3)	64.95161	22.251872	30.1619	7.22197566
GS_Strenuous	4.428571	2.87352447	4.630435	1.17953565
GS_Moderate	4.142857	3.07872887	4.717391	2.58415665
GS_Mild	6.166667	5.4459465	5.456522	3.19058645
GS_Activity_Score	79.07143	50.878602	81.63043	25.2771984
Primary_Sport				
Level_Competition	6.52381	6.62471922	7.826087	5.64601367
Number_Years	8.071429	5.70588668	5.391304	2.01672846
Months/Year	8.785714	3.28850813	9.608696	2.85623927
Sessions/Week	4.309524	2.49737958	3.521739	0.87227838
Min/Session	68.57143	26.196636	90.78947	36.2193609
ave min/session	72.97619	25.5149349	91.30435	33.8182774
min/week	336.369	287.847047	325.7609	158.209863
Intensity	3.142857	0.70962767	3.130435	0.64345289
Secondary_Sport				

Level_Competition	1.5	2.00656816	2.391304	2.03356227
Number_Years	5.275	4.29036558	3.717391	2.01599337
Months/Year	7.025	3.16425347	6.173913	4.19580065
Sessions/Week	2.725	1.08184783	2.586957	0.9001537
Min/Session	60.41667	24.0698164	68.4375	21.8874964
ave min/session	63.5	21.6491157	68.47826	20.1378648
min/week	177	101.136958	174.5652	90.1849494
Intensity	2.9	0.71818485	2.586957	0.65108605
VO2max_Abs (ml/min)	3736.079	595.638592	1842.894	458.653995
VO2max_kg (ml/kg/min)	49.65715	5.4805236	50.11247	7.3623552
VO2max_LBM	57.74942	6.3357864	58.27937	8.5396335
VO2max_MuscleVolume	62.1491	16.412253	63.14493	12.9387862
VO2max_HRmax (bpm)	194.0952	7.20350444	198	11.3179167
VO2max_RER	1.152381	0.06363213	1.053714	0.07954109
VO2max_Pmax (Watts, W)	307.8571	62.9001476	137.6818	30.1526061
VO2max_Pmax_kg (W/kg)	4.086187	0.64713592	3.764283	0.57738461
aerob_Pmax (W)	282.7222	55.1046779	130.2609	34.1390828
aerob_Pmax_kg (W/kg	3.752652	0.56051846	3.563214	0.74969951
aerob_Pmax_LBM	4.365116	0.65815858	4.143603	0.86856804
aerob_Pmax_MuscleVolume	4.689659	1.30263362	4.472098	1.08377299
EMGth_prot (sec./W)	2.714286	0.6436503	6.130435	1.25424181
EMGth_startPO (W)	102.8571	20.7708587	51.08696	11.5754954
EMGth_HRmax (bpm)	189.5	8.67240026	197.3913	8.8663774
EMGth__Finish_time (sec.)	604.4286	53.6652322	594.6522	78.0702531
EMGth_Finish_Pmax (W)	330.2381	62.3874224	150.087	36.9630197
truncated_EMGth__Finish_time (sec)	596.6381	54.1350892	578.1761	77.0517994
truncated_EMGth_Finish_Pmax (W)	328.4119	61.39294	147.9722	35.988762

EMGth_Finish_Pmax_kg (W/kg)	4.390265	0.68196317	4.045795	0.64084966
EMGth_Finish_Pmax_LBM	5.105067	0.78600525	4.719804	0.75044483
EMGth_Finish_Pmax_MuscleVolume	5.488234	1.59981299	5.088143	1.05920235
Right_Leg_EMGth (sec)	469.795	75.3581922	457.3873	87.5596777
Left_Leg_EMGth (sec)	406.15	95.5902513	491.0579	84.2405284
Comp_EMGth (sec)	412.5675	86.8357479	461.0156	87.3465576
Right_Leg_EMGth_W	281.7568	60.9314193	129.9949	30.8554317
Left_Leg_EMGth_W	254.8944	56.9200957	137.1187	31.3918095
Comp_EMGth_W	265.6774	63.0362477	129.9103	24.4176239
Comp_EMGth_VO2 (ml/min)	3565.74	691.881469	1852.898	354.77787
Right_EMGth_Wkg	3.729156	0.6752796	3.424849	0.8089567
Left_EMGth_Wkg	3.338873	0.54690625	3.559187	0.61882929
Comp_EMGth_Wkg	3.545129	0.82118087	3.557563	0.66340345
Right_Leg_EMGth_RMS (µV)	113.4485	36.3227454	76.816	28.077146
Left_Leg_EMGth_RMS (µV)	109.6811	34.2153015	74.92357	28.2481516
Comp_EMGth_RMS (µV)	108.3	38.6307299	73.17111	27.0108385
Right_EMGmax_RMS (µV)	175.2125	76.0396845	99.30667	30.8674803
Left_EMGmax_RMS (µV)	183.6878	62.778847	94.02786	39.5977587
Comp_EMGmax_RMS (µV)	175.405	65.4675128	91.94389	30.0092074
Right_EMGth_RMS_N	0.688295	0.14026353	0.774747	0.12445806
Left_EMGth_RMS_N	0.614185	0.14120106	0.815546	0.12216324
Comp_EMGth_RMS_Normalized (ratio)	0.644622	0.13991931	0.839944	0.13530066
Right_EMGth_pc_aerob_Pmax (%)	101.5081	14.4089059	97.72493	13.3616634
Left_EMGth_pc_aerob_Pmax (%)	91.15131	13.3869479	105.6768	13.5062514
Comp_EMGth_pc_aerob_Pmax (%)	94.46488	15.3338891	102.5242	13.2588719
Comp_EMGth_pc_VO2max (%)	95.66101	12.4952342	102.0767	10.776781
Comp_EMGth_Pmax (%)	80.70145	10.7379234	89.33103	10.2061342